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BILATERAL MULTIPLE OCULAR DERMOIDS

REPORT OF A CASE *

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NEW YORK

While a single ocular dermoid is not a rare observation, comparatively few cases of multiple dermoids affecting both eyes have been reported. The presence of several such growths on each eye associated with a bilateral partial ptosis makes the case reported noteworthy.

History.—M. C., a girl, aged 2 years, was first seen at Bellevue Hospital, service of Dr. John M. Wheeler, on March 21, 1927, with the history of having had growths on both eyes since birth. The parents said that there had been a slow increase in the size of those on the left eye. The family and the past history were unimportant.

Examination.—The general physical examination revealed no congenital anomalies other than those of the eyes. The examination of the eye showed, in addition to the dermoids, a bilateral partial ptosis and a moderately high hyperopia (right eye, 4 diopters; left eye, 6 diopters) as the only other pathologic observations.

In the upper part of the cornea of the right eye were three separate, bluish-gray, circular masses which were slightly elevated. These growths straddled the limbus, invading both the cornea and the conjunctiva. They were firm, almost avascular and covered by practically normal-looking epithelium. The largest growth measured 4 mm. in diameter and showed an elevation of 1 mm., while the smallest was 2 mm. in diameter and was practically flat. The rest of the cornea and conjunctiva was normal, and the eye was free from any signs of irritation..

In a similar position in the cornea of the left eye were three pinkish-yellow growths which, although larger, more elevated and more vascular than those in the right eye, were definitely of the same character. The surfaces were dry and epidermoid in contradistinction to the smooth epithelial surface covering those on the right eye. Fine downy hairs could be seen coming from all of the growths of this eye, but none from those on the right eye. The largest growth that had invaded the pupillary area measured 6 by 4 mm., the long axis being vertical and elevated about 2 mm. To the temporal side of the large growth was a smaller one, measuring about 2 mm. in diameter, which had partially fused with it; just temporal to the smaller one was still another growth, which was separated from the others by a narrow rim of corneal tissue. Examination under general anesthesia disclosed another growth between the external and superior recti, several millimeters from the limbus. This mass was yellowish, firm and oval and measured about 10 mm. in its longitudinal axis. The overlying conjunctiva, which was tightly adherent, thickened and somewhat dry, looked not unlike skin.

As the pupillary area was being invaded, it was decided that the growths should be removed from the left eye. This was accomplished in two stages; first, the

* Submitted for publication, Dec. 8, 1928.

lowermost corneal and subconjunctival growths were removed, and later the upper two corneal masses were dissected off. The resulting raw surfaces on the cornea were allowed to epithelize, and no conjunctival flaps were placed over the wounds. Convalescence was uneventful in each instance with little reaction following the operations. Nine months later the resulting corneal opacities were flat, and there was no evidence of recurrence. The right eye was not operated on, and the growths have not progressed.

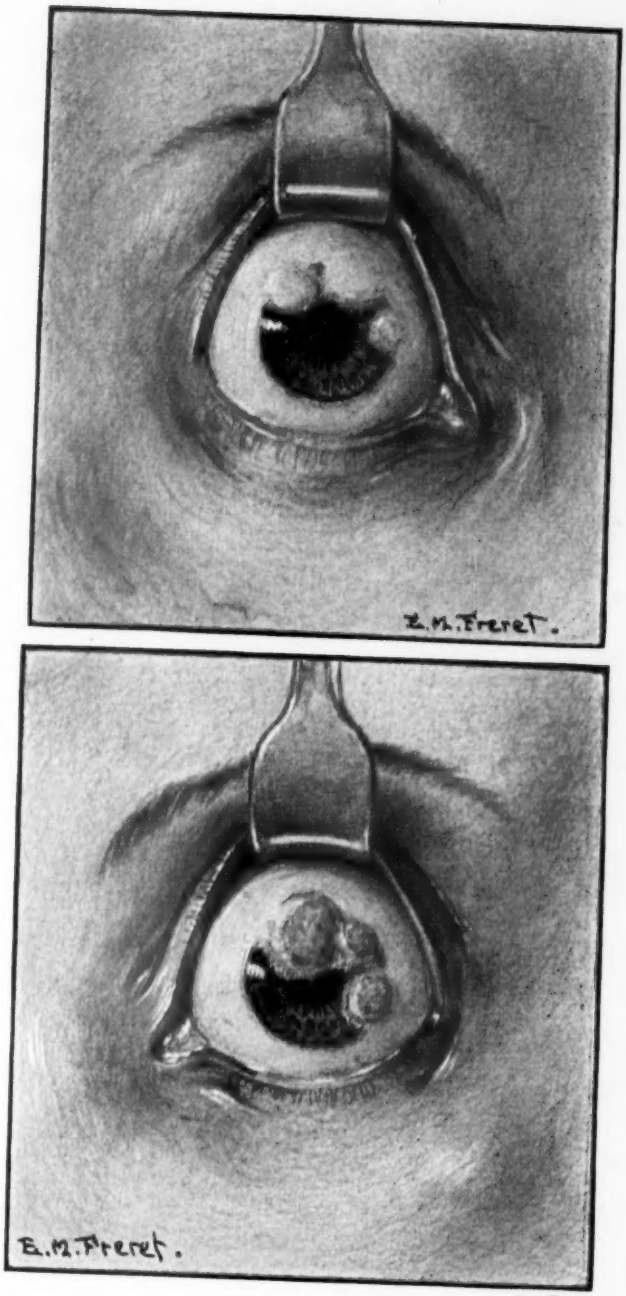
Microscopic Examination.—Microscopic examination of the tissue removed was made by Dr. Bernard Samuels, who reported as follows: "The sections submitted have the structure of skin; hair follicles and sebaceous glands being present. Deep down in the tissue there are some mucous glands. The picture is that of dermal rests in the conjunctiva."

COMMENT

The occurrence of three separate corneoscleral dermoids on each eye is, I believe, unusual, for while several cases of bilateral dermoids have been reported, most of the patients have had only one growth on each eye. Ellett [1] excised a dermoid of the conjunctiva, cornea and sclera from the left eye of a patient who had previously had a similar growth removed from the other eye. Zentmayer [2] also recorded the satisfactory removal of bilateral dermoids, his patient having two on the right and one on the left eye. The presence of three dermoids in one eye was noted by Noyes,[3] Hummelscheim,[4] Wagenmann [5] and others; however, in none of these instances was there involvement of the other eye. In 1913, Stoll [6] reported an unusual case of bilateral multiple lipodermoids with two conjunctival growths on the right eye and three conjunctival and one corneoscleral on the left eye. He considered the presence of four such growths on one eye as unique, and my search of the literature has not yielded a single other report of such a large number of separate dermoids occurring in one eye.

Several authors, among whom are Landolt,[7] Zentmayer and Collins and Mayou,[8] have commented on the tendency for bilateral dermoids to be symmetrically placed in the two eyes. The corneoscleral growths in this case show a perfect symmetry in arrangement. The location of the conjunctival dermoid between the superior and the external recti is at the favorite site, for Pignatari [9] found that 70 per cent of the growths occurred in this region. As dermoids are so frequently accompanied by other congenital anomalies, it is rather odd to find so many

1. Ellett: Memphis M. Month **29**:121, 1909.
2. Zentmayer: Tr. Am. Ophth. Soc. **27**:341, 1919.
3. Noyes, quoted by Ayres: Am. J. Ophth. **12**:289, 1895.
4. Hummelscheim: Inaug. Dis., Giessen, 1907.
5. Wagenmann: Arch. f. Ophth. **74**:511, 1910.
6. Stoll: Am. J. Ophth. **30**:1, 1913.
7. Landolt: Rep. Soc. franç. d'opth., May, 1912.
8. Collins and Mayou: Pathology and Bacteriology of the Eye, Philadelphia, P. Blakiston's Son & Company, 1925, p. 141.
9. Pignatari: Abstr., Ophth. Rev. **34**:57, 1915.



Multiple dermoids of the corneoscleral junction of both eyes (John H. Dunnington).

patients with a bilateral partial ptosis as the only other congenital defect. The presence of ptosis in these cases has been noted by Hirsch,[10] Stoll and others. The fact that the growths on the right eye are apparently covered by normal, and not epidermal, epithelium need not lead one to believe that they are different in character from those on the left eye, for Parsons[11] stated that this occasionally occurs in dermoids, especially when they are covered by the lids. The only interesting feature of the microscopic examination is the presence of an acinous gland well below the epidermal surface. De Schweinitz,[12] Parsons and others also found these glands in dermoids of the limbus.

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10. Hirsch: Klin. Monatsbl. f. Augenh. **50**:1, 1912.

11. Parsons: The Pathology of the Eye, New York, G. P. Putnam's Sons, 1904, vol. 1, part 1, p. 135.

12. De Schweinitz: Diseases of the Eye, ed. 8, Philadelphia, W. B. Saunders Company, 1921, p. 308.

TREATMENT OF PTOSIS

THE FORMATION OF A FOLD IN THE EYELID AND RESECTION OF THE LEVATOR AND TARSUS *

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In every operation for ptosis, one should, for cosmetic considerations, attempt to produce a correctly placed fold in the eyelid. In 1923,[1] I described an operation for ptosis in which I attempted to raise the eyelid by shortening the levator and by diminishing the size of the tarsus. Even in this first report I designated as an objectionable feature of this operation the fact that the fold in the lid sometimes becomes displaced toward the edge of the eyelid. Figure 1 shows that a good position of the lid can be produced with this method. In the article referred to I also described a method for subsequently correcting this undesirable displacement of the fold.

Since my operation for ptosis gave good results otherwise, and since any degree and kind of ptosis could be completely corrected by it, I attempted even in the first operation to avoid the aforementioned mistake. The displacement of the fold in the eyelid might have been caused partly by the fact that the attachments between the levator and the skin were completely divided and the latter, therefore, as a result of its weight, sank toward the edge of the lid; and, on the contrary, partly by the fact that the attachments mentioned remained undivided, and the levator pulled the skin of the lid with it when it was raised.

However that may be, the reattachment of the skin to the levator at the correct place must result in the formation of the desired fold in the eyelid. One can readily accomplish this with three interrupted sutures which are inserted during the shortening of the levator and the tarsus by the method I shall describe:

OPERATIVE PROCEDURE

The upper lid is everted by means of a lid-holder, and the operative field is anesthetized by an injection of 2 cc. of a procaine hydrochloride and epinephrine solution under the conjunctiva of the fornix. In the conjunctiva, thus stretched, one can easily make an incision along the convex border of the tarsus with a scalpel without cutting the underlying levator. If the incision is not made too near the tarsus, the wound gapes at once, and one can proceed immediately to introduce the three interrupted sutures into the proximal edge of the incision in

* Submitted for publication, Sept. 23, 1928.

1. De Blaskovics, L.: Arch. Ophth. **52**:563 (Nov.) 1923.

the conjunctiva (fig. 2). This is done in such a manner that the loops of the suture lie on the epithelial side of the conjunctiva. One now grasps the three sutures at once and pulls on them; while traction is thus exerted, the conjunctiva is easily separated from the muscle with a pair of scissors. This separation should be continued upward for quite a distance.

Fig. 1.—*A* shows a case of ptosis before operation; *B*, after operation. In *B* note the good position of the lid secured by resection of the levator and tarsus.

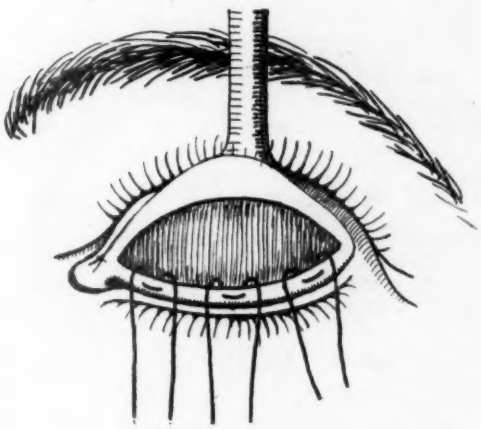

Fig. 2.—Three interrupted sutures introduced into edge of incision in the conjunctiva.

After completely exposing the levator one inserts in this muscle three fixation sutures (fig. 3). A firm bundle of the levator is seized with toothed forceps and a strong thread is passed through it and tied. The three sutures introduced are also tied. By pulling on the fixation sutures, the levator can be incised easily with scissors without injuring the fascia praetarsalis, and the levator distal to the

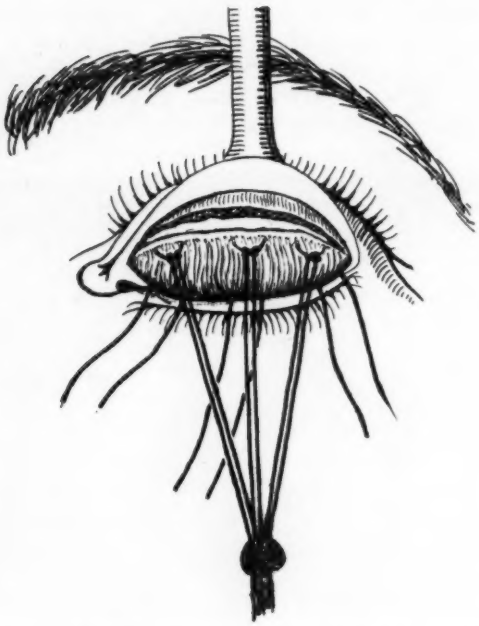

Fig. 3.—Three fixation sutures inserted into the levator muscle.

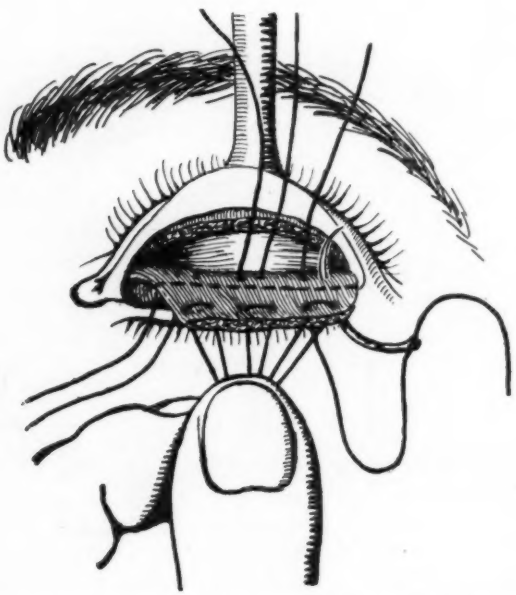

Fig. 4.—The levator muscle drawn free out of the orbit.

fixation sutures is completely divided. The muscle should be freed on both sides so that it can be pulled forward readily with the fixation sutures.

With this traction the anterior surface of the levator can also be freed, but care must be taken not to injure the fascia. The fibers of the muscle passing to the skin should also be divided. The muscle can then be drawn free out of the orbit (fig. 4). With the aid of the fixation sutures, it is now easy to pass the conjunctival sutures from behind forward at the correct height through the muscle. While pulling on the sutures, one divides the levator completely distal to the sutures introduced (the dotted line in fig. 4). A piece of the muscle is thus resected together with the fixation sutures.

The eyelid now falls back into its normal position, and the lid-holder is applied in the usual manner. One seizes the connective tissue-muscle sutures with a

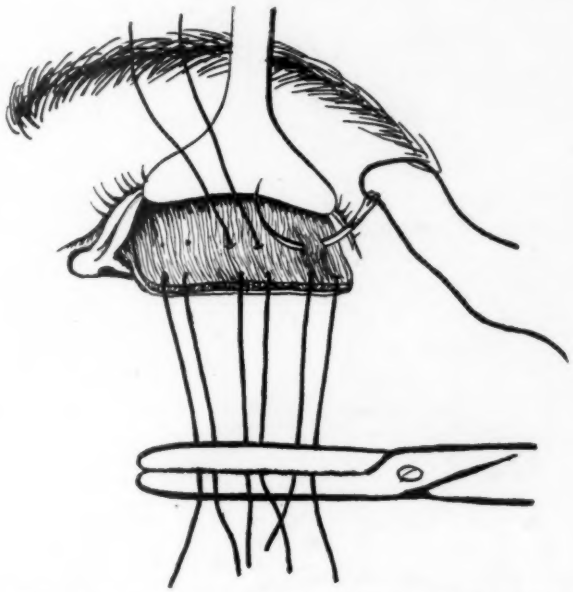

Fig. 5.—Introduction of fold-producing sutures into the levator muscle.

forceps, pulls the muscle forward and then introduces into the levator the sutures which produce the folding of the eyelid (fig. 5). These are introduced into the muscle in the same manner as the fixation sutures. They should not be placed too high in the muscle. Normally, the attachment of the levator to the skin occurs at about the level of the convex edge of the tarsus. The tarsus should be reduced, however, to about one half of its normal size. The new point of attachment should be from 4 to 5 mm. above this, so that the fold-producing sutures in the eyelid are introduced from 4 to 5 mm. above the line of incision of the muscle.

Before these can be passed through the skin, one must remove a portion of the tarsus. In order to do this one everts the lid again, seizes the stump of the muscle on the convex edge of the tarsus with a pair of forked forceps and pulls the everted lid over a lid plate inserted from below (fig. 6). In about its center the tarsus is now divided longitudinally (dotted line in fig. 6) and removed with a pair of scissors. If it seems necessary to remove more, the tarsus can be

Fig. 6.—Removal of portion of the tarsus, as indicated by the dotted line.

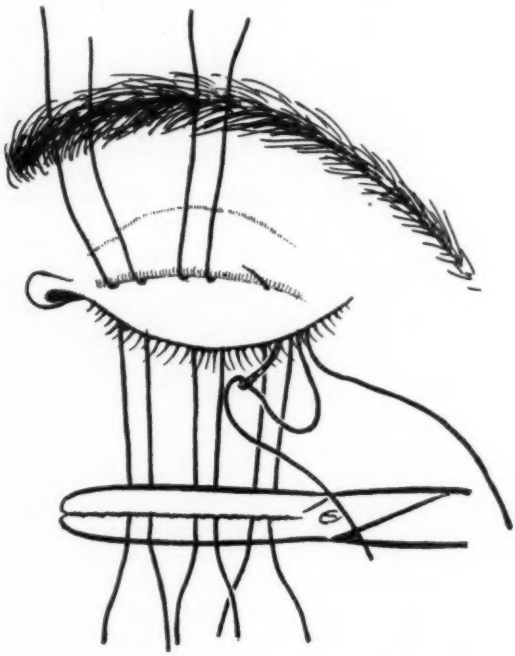

Fig. 7.—Completion of fold-forming sutures.

reduced to even 1.5 mm. A narrow strip of tarsus bends, owing to the increased upward pull of the levator, and thereby widens the palpebral fissure a great deal more than the stretching of the muscle alone could have done.

After removing a sufficiently large portion of the tarsus, one replaces the lid in its normal position and completes the fold-forming sutures. The interrupted sutures introduced into the muscle are passed through the lid from behind forward at the height at which one wishes to have the fold in the eyelid (fig. 7). At this point one usually sees fine wrinkles passing along the lid so that the level at which the sutures should be inserted can be easily determined. Even before one ties the sutures, one sees the fold appear in the eyelid. After they are tied, not too tightly, the knots disappear in the depths of the newly formed fold. One does not cut off the ends of the sutures but proceeds at once to the last step of the operation, fixation of the stump of the levator.

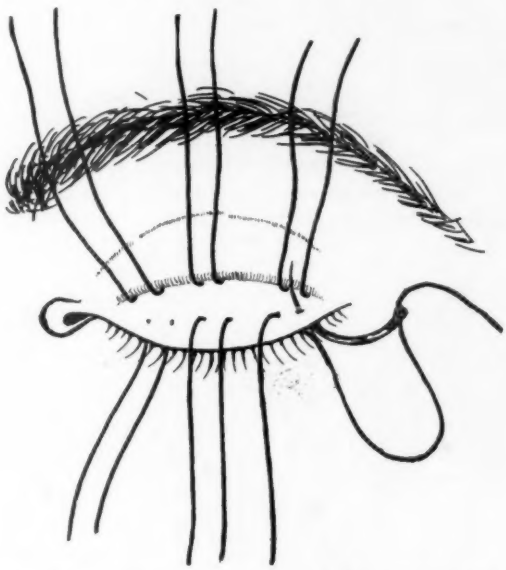

Fig. 8.—Completion of sutures in the conjunctiva.

The conjunctiva-muscle sutures still hang out of the palpebral fissure. First, the forceps on them is taken off. It is unnecessary to suture the muscle to the stump of the tarsus. The muscle unites with it just as well when the sutures are pulled close over the edge of the incised tarsus through the eyelid. This is likewise performed from behind forward (fig. 8); in doing this one should take care that the sutures are brought through the skin neither too high nor too low. In the first case the skin of the lid would be pushed toward the edge of the lid and as a result the eyelashes would come to be directed downward; in the second case, ectropion would in all probability develop. Both errors in technic can easily be avoided if one does not displace the skin of the eyelid in pushing the needle through it but instead pushes it directly through the part of the skin raised by the point of the needle.

After these sutures are tied, their ends are cut off. The sutures forming the fold in the lid, however, are preserved and are used to assure the closure of the

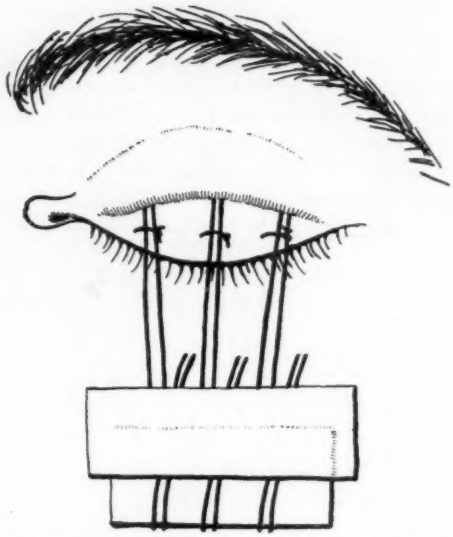

Fig. 9.—Ends of fold-forming sutures are held against the cheek by means of a band of adhesive plaster, for twenty-four hours.

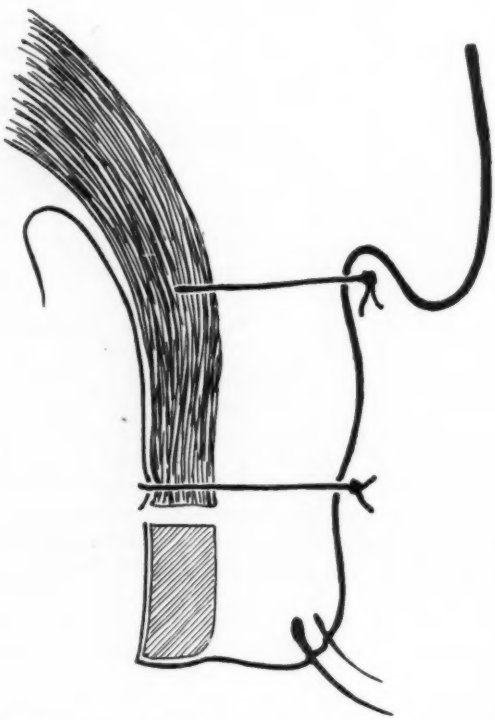

Fig. 10.—The levator muscle heals smoothly against the stump of the tarsus.

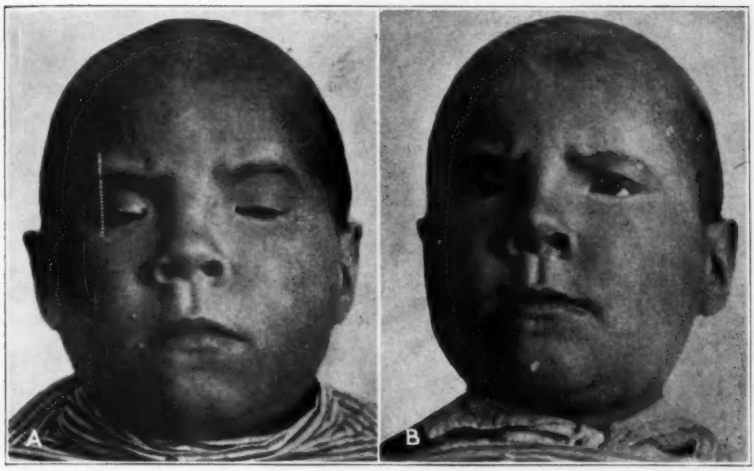

Fig. 11.—*A* shows a case of congenital ptosis before operation; *B*, the result of resection of the levator and tarsus. There is improvement in saddle nose by the transplantation of costal cartilage.

Fig. 12.—*A* shows a case of incomplete congenital ptosis; *B*, the excellent cosmetic results obtained from this operation for formation of the lid-fold. The patient's vision was improved.

eye under the bandage. For this purpose one attaches them, at the correct tension, to the cheek by means of adhesive plaster. The attachment of the ends of these sutures can be increased with a second strip of adhesive after bending them backward (fig. 9). After twenty-four hours these sutures can also be divided in the usual manner, and the adhesive plaster can be removed. From now on the eye is protected by a Fuchs shield. On the sixth day all the sutures are removed. The levator always heals smooth with the stump of the tarsus, although it was brought only close to it (fig. 10). The point of exit of the fold-forming sutures becomes the highest point of the skin fold.

COMMENT

It should be emphasized that the immediate effect is less than the effect to be expected. At the time of operation the tissues are swollen by the injection and the levator is paralyzed. Time is required for the bending of the tarsus upward. It is certain, therefore, that the immediate effect will become considerably more pronounced during the succeeding weeks; this is the opposite of what occurs when the other methods are used, by which there is a decrease in the immediate effect, as a rule. An additional advantage of the method is that the closure of the lid leaves nothing to be wished for; indeed an apparent function of the levator develops even in those cases in which this muscle was completely paralyzed or atrophic.

The movements of the lid occur in the normal direction, since the levator is the only muscle that can raise the lid in the correct manner. Now instead of the muscular contractions one finds the tension resulting from the operation, which develops as soon as the innervation of the orbicularis ceases. Even when the muscle is absent the operation can be performed and be effective. In these congenital cases the levator is replaced by a layer of connective tissue which can be used just as well as a fully developed muscle in raising the lid. Figure 11 shows the efficacy of the resection of the levator and the tarsus. In the layer of connective tissue replacing the levator, in this case, there were only scattered fibers of smooth muscle. In spite of the narrow palpebral fissure and the rough skin, a sufficiently wide palpebral fissure and complete closure of the lids were obtained by decreasing the tarsus considerably and shortening the levator as much as possible. The saddle nose in this case was improved by the transplantation of costal cartilage, according to Lewis.

In all of twelve cases of ptosis in which I used the lid fold-forming operation the results were excellent. Figure 12 demonstrates the cosmetic effect of such an operation in a case of incomplete congenital ptosis. The patient nevertheless uses her frontalis muscle constantly. Although a fold in the lid was noticeable even before the operation, the patient's vision afterward became much better when a normal appearing and correctly placed lid-fold had developed.

THE ARTIFICIAL INDUCTION OF OCULAR HYPERTENSION BY COMPRESSION OF THE JUGULAR VEINS

ITS PHYSIOLOGIC ASPECT: AN INTERPRETATION *

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NEW YORK

About a year ago, one of my associates at the Knapp Memorial Eye Hospital asked me to watch a very "unusual occurrence." He applied the Schiötz tonometer to one of the eyes of a patient suspected of being glaucomatous, and it indicated six divisions on the tonometer scale. (The weight used was 7.5) Within a few seconds, the indicator moved to division 5 and then to 4. He could not understand the meaning of this hardening of the eyeball under the weight of the tonometer constantly applied to it when he knew that usually the opposite (a softening) happens under such circumstances. A glance at the patient's face gave a satisfactory explanation. The patient was sitting with his head bent far back so as to have the eyes look straight up; his collar, tightly stretched around his neck, exerted a mild strangulation of the jugular veins, and the face was cyanotic. Under such circumstances a venous engorgement of the entire head and of course the eyeballs must have taken place, hence the increase in ocular tension. The admonition was given: "Use the tonometer on the patient in a lying position or at least without a tight collar around his neck." A few months later, the same occurrence was experienced by another of my associates. Thus, the question as to whether the physiologic and pathologic variations of intraocular pressure have anything to do with the venous system in or immediately outside the eyeball began to preoccupy me. Queckensted has shown that compression of one or both jugular veins raises the pressure of the cerebrospinal fluid from 8 mm. of mercury (normal) to 16 mm. if one jugular vein is compressed and to 24 mm. if both are compressed. The rise takes place within from about five to thirty seconds of the jugular compression, and if this is continued for some time the return flow of blood from the brain is almost entirely interfered with and serious disturbances appear (vertigo, headache, tremor and convulsions).

Claude, Lamache and Aubry [1] confirmed Queckensted's experience, and found that the increase of pressure of the cerebrospinal fluid is

* Submitted for publication, Jan. 2, 1929.

1. Claude, H.; Lamache, A., and Aubry, M.: Compt. rend. Soc. de biol. **96**:261, 1927.

gradual and that the release of jugular compression is followed by a return to the initial figure in from ten to fifteen seconds. The increase was greater in young, vigorous people than in persons with an "inert vegetative system." These authors attributed this rise to a mechanical factor—venous stasis produced by compression of the jugular veins. The following case is a plausible argument in favor of this conception: In a patient with thrombosis of one of the jugular veins, compression of the jugular on the side on which thrombosis was present did not cause an increase in pressure of the cerebrospinal fluid, while compression on the healthy side did. However, the authors think that there is another contributory factor in addition to the mechanical factor, a reflex due to the excitability of the neurovegetative system.

In 1906, Wessely [2] reported that compression of the neck of rabbits often produces an increase of intra-ocular pressure, but that as the compression is continued the intra-ocular pressure gradually drops. In 1907, Ernst Schulze [3] published his paper on the influence of compression of the neck on intra-ocular pressure. He applied a rubber band around the neck of rabbits for two hours and with a Fick-Livschitz tonometer studied the effect on intra-ocular pressure. He found that the ocular tension increased from 18 to 35 mm. after two minutes; then it fell to 25 mm. four minutes later and to 22 mm. two minutes afterward. The release of the neck band brought the ocular tension to 10, 8 and finally to 6 mm. The author attributed the gradual fall of the tension after the initial rise to a squeezing out of the fluids from the eyeball. In a few rabbits, the band was kept applied around the neck (not so tightly stretched) for several days. In these animals, the tension did not rise as much as in the first series. Schulze also tried the compression of the neck with a rubber band on two patients. In the first patient the tonometer rose from 28 to 34 after five minutes and then fell to 22 two minutes later. In the second patient, the tonometer rose from 20 to 22. Schirmer, discussing Schulze's paper, remarked that he tried the Bier hyperemia method by applying a rubber band around the patient's neck in various conditions, but that he never found an increase of intra-ocular pressure. Thiel [4] measured the ocular tension in patients in a reclining position. The table was tilted from 10 to 30 degrees so that the head and chest were lower than the rest of the body. Under such conditions he found that the ocular tension rises about 15 mm. in patients with glaucoma, and he attributed it to the engorgement of the veins of the head. Thiel compressed the jugular veins with a specially constructed apparatus, consisting of a band of metal

2. Wessely: Ber. Heidelberg Versammlung der deutsch. ophthalm. Gesellsch., 1906.

3. Schulze, Ernst: Ztschr. f. Augenh. **17**:222, 1907.

4. Thiel: Berlin Fortbildungskurs für Augenaertze, 1925, p. 80.

called "Vasco." After an application of this apparatus for from one to two and a half hours, the face becomes congested and the episcleral vessels are engorged. The healthy eyes regain their normal tension soon after the beginning of the pressure on the neck. Glaucomatous patients, however, apparently on account of the insufficiency of the "regulatory apparatus," register an increase of tension which lasts as long as the compression is applied to the neck.

It should be noted that all these authors used a crude method of compression of the neck and were unable to know how much pressure they applied. Also, the tonometer used, except that of Thiel, was neither practical nor as reliable as the Schiötz or the Maclane tonometers. Bonnefon [5] repeated and confirmed the experiments of Schulze. Comberg and Stoewer [6] studied the effect of muscular contractions of various regions of the body on the intra-ocular pressure. They said that even lay people know that "the blood rushes to the head" during a sudden strong contraction of muscles of the chest and abdomen and that the eyes become red and even bulging during this effort. The authors measured the blood pressure on the arm during a forced muscular contraction of the chest and abdomen and found an increase of from 25 to 35 mm. systolic and from 30 to 40 mm. diastolic. Physiologists consider that the muscular contraction of the chest compresses the lungs, interferes with the circulation of the blood in this organ and causes a stagnation in the venous system (Valsalva experiment). This explains the increase of blood pressure in Comberg's experiment. At the same time the ocular tension was also measured, and it was found that it increased in these patients from 18 to 30, from 18 to 29 and from 18 to 37 mm. and that the eyeballs protruded slightly, about 0.5 or 0.75 mm. Hiroishi [6a] found that isotonic suspension or fine carbon particles introduced into the anterior chamber (in rabbits) will appear in Schlemm's canal and the episcleral vessels when the pressure in the anterior chamber was from 20 to 25 mm. and that pressure on the jugular veins stopped the drainage of these carbon particles from the anterior chamber.

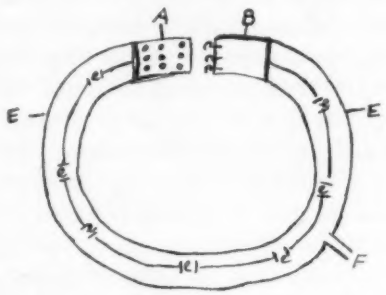
This is about all that is found in the ophthalmologic literature that may be relevant to the subject of the direct relation between the venous system and intra-ocular pressure.

My purpose in the investigation to be reported in this paper was to devise a simple method whereby a definite amount of pressure, measured in millimeters of mercury, was to be exerted on the jugular veins and the effect of such pressure on the records of intra-ocular tension.

5. Bonnefon: Ann. d'ocul. **159**:159, 1922.
6. Comberg, W., and Stoewer, E.: Ztschr. f. Augenh. **58**:92, 1925.
6a. Hiroishi: Arch. f. Ophth. **113**:212, 1924.

METHOD OF COMPRESSION OF THE JUGULAR VEINS

It was found that an ordinary Tycos blood pressure apparatus answers the purpose. The cuff is applied around the patient's neck, and air is pumped until the pressure meter shows 40 mm. of mercury. The ocular tension is measured before and as soon as the air is pumped in the cuff. Usually there is an increase of 1 or 2 divisions on the tonometric scale (when a 7.5 weight is used) in both eyes after the inflation of the cuff. If the patient is healthy and does not show any signs of anxiety and if the ocular tension does not change, more air may be pumped up to 60 and even 80 mm. If, on the other hand, the tonometric scale shows a rise of one division, already at 20 or 30 pressure, it is not necessary to pump in more air. The purpose is to determine the minimum pressure necessary to raise the ocular tension just one division on the tonometric scale. Another tonometric measurement is taken after the blood pressure apparatus is removed. Thus three tonometric measurements are obtained: the first before pressure is applied on the neck; the second during the application of the pressure, and the third after the release of pressure. Usually the eye becomes harder during the application of pressure to the neck and softer after the release of pressure. The third measurement after the release of pressure shows a tension even lower than the initial one before the pressure on the neck was applied. When the air is pumped into the cuff, it presses with equal force all around the neck and, of course, on the larynx, causing a feeling of anxiety in some patients. To avoid this, a special cuff was designed, which is constructed in such a manner as to press only on the sides of the neck in the region of the jugular veins, leaving the larynx entirely free. This cuff consists of two flat tubes made of rubber tissue, one alongside the other and communicating by openings with each other. They form a broad neck band which hooks up in front of the larynx. When the air is pumped into this band (by the aid of a Tycos apparatus) it is inflated on each side but not in front of the larynx. The principle of its construction is that of a pneumatic horseshoe (*E-E*) with the opening in front of the larynx, bridged over by a connecting strap (*A-B*) and provided with hooks and several rows of holes for adjusting the band to the various sizes of the neck. As the tube is being inflated, two effects are produced: (1) compression of the sides and back of the neck and (2) tendency of the tube to straighten out. Thus, the larynx is spared from compression.



Cuff used in tonometric measurement. *A-B*, connecting strap; *E-E*, pneumatic horseshoe; *e-e*, communications between the two flat tubes; *F*, tube to be attached to Tycos apparatus.

I am indebted to my colleague, Dr. Joshua Rosett of New York, for the construction of this cuff. It works well and causes little, if any, feeling of anxiety.

A proper examination cannot be made without an assistant. The patient is lying comfortably, preferably on a table with the head slightly elevated. The ocular tension is measured, then the cuff is applied around the neck with the noninflatable part over the larynx. The assistant pumps in the air with one hand, gently pulls down the lower lid of the eye to be tonometrized with the other hand and watches the meter so as to keep the pressure constantly at one level during the examination. The examiner gently retracts the upper lid with one hand and applies the tonometer with the other hand. Only by adhering strictly to the technic is one able to obtain reliable data.

CASES ILLUSTRATING SOME OF THE REACTIONS OF THE OCULAR TENSION TO PRESSURE ON THE JUGULAR VEINS

The weight used in the tonometer was 7.5, and for the sake of simplicity the measurement of the ocular tension is expressed in divisions on the Schiötz scale instead of in millimeters of mercury.

I. Normal Eyes

Case 1 F, a woman, aged 59.

Ocular tension, right eye, 7 / left eye, 7 } pressure on jugulars 60 mm. of mercury. Tension { Right eye, 5½ / Left eye, 7

Case 2 F, a woman, aged 39.

Ocular tension, right eye, 7 / left eye, 7 } pressure on jugulars 30 mm. of mercury. Tension { Right eye, 6 / Left eye, 6

Release of pressure, right eye, 7½
left eye, 7½

II. Cases of Cataracta Senilis

Case 3 E, a patient, aged 68, incipient cataract, right eye.

Ocular tension, right eye, 8; pressure on jugulars, 40 mm. of mercury. Tension, right eye, 7
After release of pressure, tension, right eye, 8

Case 5 E, a patient, aged 63; incipient cataract, each eye.

Ocular tension, right eye, 6 / left eye, 6 } pressure on jugulars 40 mm. of mercury. Tension { Right eye, 4½ / Left eye, 5

Case 6 E, a patient, aged 56; mature cataract, right eye; aphakia, left eye.

Ocular tension, right eye, 7 / left eye, 7 } pressure on jugular 50 mm. of mercury. Tension { Right eye, 7 / Left eye, 7

Raising pressure to 60 mm. of mercury. Tension, right eye, 5; left eye, 5
Releasing jugular pressure.....right eye, 7; left eye, 6

III. Cases of Uveitis

Case 1 D, a man, aged 58.

Ocular tension, right eye, 6 / left eye, 6 } pressure on jugulars 20 mm. of mercury. Tension { Right eye, 4 / Left eye, 4

Release pressure, tension, right eye, 6
left eye, 6

Same patient, second examination.

Ocular tension, right eye, 6½ / left eye, 6½ } forced expiration.....Ocular tension { Right eye, 5 / Left eye, 6

Case 2 D, a man, aged 56; acute iridocyclitis; secondary glaucoma (bilateral).

Ocular tension, right eye, 4 / left eye, 2½ } jugular pressure 20 mm. of mercury. Tension { Right eye, 4 / Left eye, 2½

Release of pressure, tension, right eye, 4½
left eye, 2½

IV. Cases of High Myopia with Vitreous Degeneration

Case 1 C, a woman, aged 61; low blood pressure.

Ocular tension, right eye, 8 / left eye, 8 } jugular pressure 50 mm. of mercury.....Tension { Right eye, 11 / Left eye, 11

As one sees in this case, the ocular tension decreased.

Case 2 C, a woman, aged 48; old healed iritis in left eye.

Ocular tension, right eye, 6½ / left eye, 7 } jugular pressure 40 mm. of mercury. Tension { Right eye, 5 / Left eye, 6

Release of pressure, tension, right eye, 6
left eye, 6

Case 3 C, a woman, aged 40; high blood pressure.

Ocular tension, right eye, 5½ / left eye, 5½ } jugular pressure 40 mm. of mercury. Tension { Right eye, 3½ / Left eye, 4½

Release of pressure, tension, right eye, 6½
left eye, 7½

Case 5 C, a girl, aged 18; high myopia in right eye; moderate myopia in left.

Ocular tension, right eye, 8 / left eye, 8½ } jugular pressure 40 mm. of mercury. Tension { Right eye, 7 / Left eye, 8

Release of pressure, tension, right eye, 9
left eye, 9

Case 7 C, a woman, aged 40.

Ocular tension, right eye, 7 / left eye, 7 } jugular pressure 40 mm. of mercury.....Tension { Right eye, 5 / Left eye, 5

Release of jugular pressure, tension, right eye, 8
left eye, 8

V. Cases of Primary Glaucoma

Case 1, a woman, aged 53; Lagrange's operation, right eye; left eye, trephined.

Ocular tension, right eye, 7 / left eye, 5 } jugular pressure 40 mm. of mercury.....Tension { Right eye, 5½ / Left eye, 4

Release of pressure, tension, right eye, 7
left eye, 5½

Case 2, a woman, aged 60; acute glaucoma, right eye; simple gloucoma—pilocarpine, left eye.

Ocular tension, right eye, 2½ / left eye, 4 } jugular pressure 40 mm. of mercury. Tension { Right eye, 2½ / Left eye, 4

Release of pressure, tension, right eye, 2½
left eye, 4

Case 3, a woman, aged 52; acute glaucoma—iridectomy, right eye; simple glaucoma, left eye.

Ocular tension, right eye, 4½ / left eye, 5 } jugular pressure, 40 mm. of mercury; tension 4; release, right eye, 4½; left eye, 5

Case 5, a woman, aged 40; acute glaucoma, right eye; simple glaucoma, left eye; each eye trephined.

Ocular tension, right eye, 6 / left eye, 5 } jugular pressure 20 mm. of mercury.....Tension { Right eye, 5 / Left eye, 5

Release of pressure, tension, right eye, 6
left eye, 6

Case 7, a woman, aged 66; simple glaucoma, each eye; iridectomy, each eye.

Ocular tension, left eye, 7, jugular pressure 40 mm. of mercury.....Tension, left, eye, 7

Release of pressure, tension, left eye, 8

Case 8, a man, aged 50; simple glaucoma, right eye; subacute glaucoma, left eye; iridectomy, each eye.

Ocular tension, right eye, 3½ / left eye, 5½ } jugular pressure 20 mm. of mercury. Tension { Right eye, 2 / Left eye, 4

Release of pressure, tension, right eye, 3
left eye, 5½

Case 9, a man, aged 52; simple glaucoma, each eye.

Ocular tension, right eye, 5½; jugular pressure 40.....Tension, right eye, 4½

Ocular tension, lett eye, 5½; jugular pressure, 40 mm. of mercury.....Tension, 4½

Release of pressure, tension, right eye, 7
left eye, 7

VI. Cases of Secondary Glaucoma After Parenchymatous Keratitis

Case 1 A, a woman, aged 26; two years after the acute stage had subsided.

Ocular tension, right eye, 6 / left eye, 4 } jugular pressure, 30 mm. of mercury.....Tension { Right eye, 4½ / Left eye, 1

Release of pressure, tension, right eye, 5½
left eye, 2

Case 2 A, a man, aged 29; five years after the subacute stage subsided.

Ocular tension, right eye, 6 / left eye, 6 } jugular pressure, 30 mm. of mercury.....Tension { Right eye, 5 / Left eye, 5

Release of pressure, tension, right eye 6
left eye, 6

COMMENT

In all these cases, both normal and pathologic, there are three distinct types of curves: in *A*, ocular tension is increased during jugular pressure and decreased to the initial figure after the release of pressure; in *B*, ocular tension is increased during pressure and decreased below the initial figure after the release of pressure; in *C*, there is no change of ocular tension by pressure on the jugular veins. To these three main types may be added a number of other subtypes not necessary to mention now.

It would be important to know how the pressure on the jugular veins causes an increase of ocular tension and why the release of pressure is followed by a drop, in some to the original ocular tension and in others even to a lower level.

Let us consider the anatomic and physiologic fundamentals of this problem. The bulk of the blood entering the head is drained off by four veins: one internal and one external jugular on each side of the neck. A small portion returns via the suboccipital plexus into the vertebral and deep cervical veins and from there into the innominate vein. A moderate amount of constriction (from 20 to 80 mm. of mercury) applied around the neck may be of sufficient intensity to interfere with the venous circulation, without affecting an appreciable amount of the arterial circulation. It is well known that interference with the return circulation of an organ has the immediate effect of tending to increase its volume and, consequently, the pressure within the shell or capsule containing this organ. The eyeball and the contents of the skull are eminently prone to a stagnation of their venous blood whenever the circulation in their main veins is more or less obstructed.

Physiologists have established the fact that there are a number of agents which promote the circulation in the veins. One factor is the muscular contractions; another factor is gravity, for the venous circulation above the level of the heart; the third is the suction exerted on the heart and larger thoracic veins during respiration, and the fourth is the action of the right side of the heart. The venous circulation of the head is promoted by all of these agents and may be disturbed whenever any one of them is handicapped. Gravity as a factor may be lessened when a person's occupation is such as to compel him to work with his head lowered or when, on account of illness, the body is in a horizontal position. The contractions of the muscles of the neck running alongside or across the course of the jugular veins undoubtedly promote or retard the return flow of the blood in the veins. Finally, the state of normality or abnormality of the organs of the chest (mainly of the lungs and heart) must also have a certain influence on the ease with which the contents of the jugular veins empty into the right side of the heart. All these factors are not to be ignored, and it is time that more attention should be paid to them whenever the question of intra-ocular pressure is being considered. But this is not all. Ophthalmologists stand little chance of understanding the whys and hows of normal and pathologic variations of intra-ocular pressure if they fail to accept as one unit the three links of the vascular chain: (*a*) arterial, (*b*) capillary and (*c*) venous. For the sake of simplicity let us call it the V.C.A. unit (venules, arterioles and capillaries). *a* has been more or less considered by ophthalmologists, *b* little, and *c* not at all. Hooker [7]

7. Hooker, D. R.: Physiol. Rev. **1**:112, 1921.

has published a summary of the evidence of functional activity on the part of the capillaries and venules. It seems that during the fifty years of research following Claude Bernard's discovery of vasomotor action, a number of important facts have been brought out concerning the independent function of the arteriocapillaries and the venocapillaries. The venous side may act more or less independently of the arterial side of the vascular system. There are a number of reasons (anatomic, physiologic and pharmacologic) that make probable the belief that the capillaries and venules have their own independent function. Steinach and Kahn, using faradic or galvanic stimuli, obtained a contraction of the lumen of the capillaries as well as of the venules. They found that in venules the contraction is less energetic; it develops more slowly and seldom reaches the degree of a complete obstruction of the lumen. The period of relaxation lasts a shorter time in the venules than in the capillaries. These differences of contraction between the venules and the arterioles are ascribed to the fact that there are fewer contractile elements in the walls of venules. Capillaries and venules contract by direct stimulation of the tissues or by stimulation of the nerve trunk going to the tissues. Up to the present time little work has been done to determine whether the veins promote the circulation of the blood by their own independent contractions. There is abundant evidence that the tone of the veins is an important factor in circulatory regulation. Cownet,[8] Gunn and Chavasse [9] have shown that application of epinephrine on the inferior vena cava at a considerable distance from the heart elicits rhythmic contractions of the vein. According to Morat and Doyon,[10] in rabbits small veins of the ear contract spontaneously. Years ago, I often found that when I tried to inject a large quantity of fluid into the veins of the ear in rabbits the fluid would enter with great facility if the animal was at ease. When the rabbit was agitated, it was impossible to inject any fluid because it seemed as if the vein was closed farther down. Briscoe [11] concluded from his observations on venous and capillary pressure that capillary stasis (with high capillary pressure and normal venous pressure in an irritable heart) with cyanosis of the hands is due to a constriction of the venules. Hooker showed experimentally that a central as well as a peripheral venopressor mechanism exists. Heubner found that double chlorides of gold and sodium produce prostration with a remarkable engorgement and stasis of the capillaries and veins. These chlorides are an endothelial poison primarily for the capillaries and veins. Dale has shown histamine to be like a double chloride, a poison to the capillaries and venules.

8. Cownet: Am. J. Physiol. **54**:96, 1920.
9. Chavasse: Proc. Roy. Soc. Lond. **86B**:192, 1913.
10. Morat and Doyon: Traite de physiologie, Paris, Masson et Cie, 1918, vol. 3.
11. Briscoe: Heart **7**:35, 1918.

It is fairly well established that chemical factors mediate dilatation of the capillaries and venules, while nerve stimulation mediates constriction of these vessels. Both these factors have long been recognized as participating in vascular reactions, but only lately have they been located in their effects on the capillaries and venules, that is, on that section of the vascular bed composed of bare endothelium (Hooker). The same author said: "Hitherto the functional peripheral resistance has been believed to be limited to the arterioles, subject to nervous and chemical influences. But now we know that beyond the arterioles, the capillaries and venules function actively and must therefore participate directly in vascular reactions."

When ophthalmologists begin to apply the data supplied by physiology to their own field, especially to the question of intra-ocular pressure, an interesting vista opens. One can produce mechanically a venous hyperemia in the brain and eyes by pressure on the jugular veins. It is probable that the venules have contractile functions similar, if less active, to those of the capillaries. Is it not possible that certain factors (mechanical, chemical, vasomotor) may produce a sudden or slow contraction of these venules and change the intra-ocular pressure in the same manner that a simple compression of the jugular veins does?

In his excellent book on the newer points of view on glaucoma, Elder [12] said "that the intra-ocular pressure depends on the capillary pressure, while the systemic high blood pressure is frequently accompanied by a low capillary pressure." There is in the normal person a regulating mechanism keeping the systemic blood pressure at a constant level, but in patients with high blood pressure, in whom this mechanism is not adequate, the pressure is subject to sudden variations (McWilliam [13]). Besides this lack of efficiency of the vasomotor system, a second factor plays an important rôle in the causation of ocular hyperemia, namely, variations in the venous pressure. The level of intra-ocular pressure depends more on the variations in venous pressure than on the variations in arterial pressure. Local variations in the eyeball within the venous return may readily give rise to a glaucomatous attack. Finally, the capillary pressure has its own independent variations, due to physical, nervous, chemical, hormonal and psychic factors. Let us return to the previous question, "How does pressure on the jugulars cause an increase of intra-ocular tension?" There is little doubt that a mechanical factor is involved in this reaction, a damming up of the venous exit from the eyeball and a rapid accumulation of blood in the uvea. But this mechanical element can hardly be the only reason. If it were, it would

12. Elder, W. V. D.: Recent Advances in Ophthalmology, Philadelphia, P. Blakiston's Son & Company, 1927, p. 343.

13. McWilliam: Physiol. Rev. **5**:303, 1925.

be hard to understand why the ocular tension drops at times below the initial figure after the release of pressure on the neck. Furthermore, why is it that, even during the continuous application of jugular compression, the initial rise of the ocular tension is followed by a subsequent fall? Something must happen to that important vascular unit within the eyeball (the venule, capillary, arteriole unit); there must exist a regulatory reflex (a defensive mechanism) as a response to that acute impediment of venous circulation. It is easy to assume that an induced distention of the venous system of the eyeball opens paths of drainage clogged up by pigment, cells and colloidal substances and that a sudden release of the jugular compression exerts a suction on these elements, thereby facilitating their removal. But if this were so, why should a drop below the initial ocular tension follow in normal eyes, in which such substances are not present in the paths of drainage from the eyeball? The problem is by far more complex than it appears on the surface. It seems to me plausible that the three links of the V.C.A. unit (venules, capillaries, arterioles) are interrelated by a coordinating mechanism, consisting of a reflex that originates in each one of them and causes a response in the other two. The result of this coordination would be that a spasm of the arterioles would reflexly contract the venules, preventing a sudden starvation of the tissues by a complete emptying of the capillary bed; a spasm of the venules would reflexly contract the arterioles so as to prevent a too sudden accumulation of blood in the capillaries. One could not understand a smooth function of the capillary circulation without these safeguards. On the other hand, a breakdown of these reflexes must have serious consequences to the nutrition of the tissues and to the evenness of capillary circulation, particularly to the maintenance of the normal level of intra-ocular tension.

SUMMARY

It would be premature to make suggestions as to diagnostic or therapeutic indications that may be derived from the foregoing observations and interpretations. The number of patients examined is small. It will be necessary to accumulate data on several thousand patients before one can venture to draw conclusions.

One thing is certain: the intra-ocular pressure is altered under the influence of compression of the jugular veins. The amount of pressure can be carefully gaged and measured in millimeters of mercury just as the blood pressure is measured. It is also probable that the change of ocular tension is due not only to mechanical stretching of the jugular veins but to a complex reflex that reacts on the capillaries and arterioles and that this reaction is not the same in all persons, not even always in the same eye. This apparent inconsistency depends on two

main factors: (*a*) the anatomic peculiarities (size, thickness and elasticity) of the walls of the veins and (*b*) the readiness with which the V.C.A. unit responds reflexly to the distention of the jugular veins.

It is the study of this reflex in the V.C.A. unit, first to such coarse stimuli as the jugular compression and later, to the more subtle stimuli (hormonic, physical, chemical reflex) that will bring about a better understanding of the normal and pathologic variations of the intraocular pressure.

Some of the factors predisposing to a weakening or stretching of the walls of the tributaries of the jugular veins are constipation, with its daily efforts at defecation which bring about marked venous congestion of the head; childbirth, certain occupations (glassblowing, the blowing of musical instruments, hard labor consisting of lifting and carrying heavy objects, acrobatic performances, scrubbing floors, etc.) faulty diets (abuse of the use of strong tea, coffee and alcoholic beverages) and infectious diseases. However, the fact that out of millions of persons subject to these predisposing factors only a minute percentage is affected with glaucoma points to the fallacy of believing that these factors are of anything but secondary importance to the normal person. They acquire a certain significance only in those in whom a conspiracy of several other reasons originates a disturbance of the regulating mechanism of the ocular tension, and this mechanism has something to do with the reflexes correlating the function of the V.C.A. unit.

THE LIGHT SENSE IN EARLY GLAUCOMA

THE SMALLEST DIFFERENCE IN BRIGHTNESS PERCEPTIBLE TO THE LIGHT ADAPTED EYE (LIGHT DIFFERENCE) *

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While continuing the work on the light minimum, we investigated the ability of the eye to detect a difference in the degree of brightness, to discover whether this might be significant as a diagnostic sign in early glaucoma. If such a test proved to be of value, it might serve as an adjunct to, or a substitute for, the light minimum test which requires a prolonged stay in the dark room and a number of readings during the course of dark adaptation.

REVIEW OF THE LITERATURE

An earlier article [1] gave a brief summary of work previously done on the light sense which seemed particularly relevant to our line of investigation. In the past few years, more work has been done which should be mentioned here. This review of the literature, like the previous one, discloses many interesting points on different phases of the light sense; however, we have been unable to find any investigations that exactly duplicate ours in aim and, at the same time—so it seems to us—in accuracy of the control of so many of the variable factors that influence a test of the light sense. Some of these factors might be enumerated:

1. Intensity of the light at the retina, which requires an accurate knowledge of the area of the pupillary opening.
2. Extensity of the light, i. e., visual angle subtended on the retina.
3. Wave length of light.
4. Time of exposure.
5. Surrounding field—simultaneous contrast.
6. The eye, region of the retina involved (center or periphery).
7. Previous condition of the eye—state of adaptation. Pre-exposure.

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1. Waite, J. H.; Derby, G. S., and Kirk, E. B.: The Light Sense in Early Glaucoma, Particularly the Achromatic Scotopic Threshold at the Macula, Tr. Ophth. Soc. **45**:301, 1925.

We believe that we have standardized some of these factors at a constant value, and that we have accurate means of controlling and measuring the others. Moreover, we calibrated on normal observers our whole arrangement of apparatus and technic.

Other work that we have seen on the light sense ignores or controls inadequately one or more of these factors. The factor most commonly omitted is the area of the pupil, although a few investigators take it into account. Hecht,[2] in carrying out further accurately controlled work on the photochemical processes in the retina responsible for discrimination of intensity differences, corrected his results by means of Reeves'[3] data on the size of the pupil, which were obtained by photographing the pupil during the course of dark adaptation. A slight source of error consists in the fact that one often finds varying degrees of ectropion uveae which would appear in the picture as part of the pupil itself. Another fact not taken into account is the variation in the size of the pupil with accommodation—some of Hecht's work was done at less than a meter. We still believe that fixation of the patient's pupils with pilocarpine or some other drug provides the easiest and most certain method of control. Hecht found, as did earlier investigators,[4] that the least perceptible light difference is approximately 2 per cent in the range of ordinary illuminations. He did no work with pathologic cases.

The only other investigator we found who recognized the action of the pupil was Möller, who belonged to the group working at Copenhagen with Tscherning's photometric glasses—a series of neutral glasses transmitting light in quantities varying logarithmically, i. e., 0.1, 0.01, 0.001, etc. The most prolific writers in this group[5] were Möller and

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2. Hecht, Selig: Visual Discrimination of Intensity and the Weber-Fechner Law, J. General Physiol. **7**:235, 1924-1925.

3. Reeves, Prentice: Rate of Pupillary Dilatation and Contraction, Psychoanalyt. Rev. **35**:330, 1918.

4. Aubert, H.: Physiologie der Netzhaut, Breslau, 1865. König, A., and Brodhun, E.: Experimentelle Untersuchungen über die psychophysiche Fundamentalform in Bezug auf den Gesichtssinn, Sitzungsb. d. Berl. Akad., 1889, p. 641. Blanchard, Julian: The Brightness Sensibility of the Retina, Physiol. Rev. **11**: 81, 1918.

5. Möller, H. U.: Ueber die Adaptation und ihre Messung durch die photometrischer Gläser nach Tscherning, Acta ophth. **1**:324, 1923; Dark Vision in Glaucoma, ibid. **3**:1 and 170, 1925; Ueber die Messung der Desadaptation mit Tscherning's photometrischen Gläsern **3**:3 and 272, 1926. Edmund, Carsten, and Möller, H. U.: Vision in Light of Reduced Intensity, Arch. Ophth. **54**:531 (Nov.) 1925. Edmund, Carsten: The Dependence of the Light Sense on the Adaptation of the Eye, Acta ophth. **2**:125, 1924; Ueber Hemeralopia idiopathica mit besonderem Hinblick auf Untersuchung und Behandlung, ibid. **2**:225, 1924; Some Methods of Testing Dark Vision, ibid. **3**:1 and 153, 1925. Frandsen, Helga: Investigations of the Range and Velocity of Adaptation by Means of Professor Tscherning's Photometric Glasses, ibid. **2**:239, 1924.

Edmund. They investigated the course of dark adaptation, the course of light adaptation, the relation of adaptation to pre-exposure, the effect of reduced illumination on acuity and on visual fields, for both normal subjects and patients with glaucoma (but usually fairly advanced glaucoma), idiopathic hemeralopia, retinitis pigmentosa and other pathologic conditions. They diminished the illumination with the Tscherning glasses, but they made no attempt to standardize their original illumination. Their preexposure consisted for the most part in "bright daylight," "diffuse evening light," etc. They took no account of the pupil, although Möller [6] remarked that the size of the pupil affects the amount of light entering the eye. He believed, however, that this would not account for the severer degrees of hemeralopia, because the difference between a 6 mm. pupil and a 2 mm. pupil corresponds to about the difference between one Tscherning photometric glass and the next in the series, which he considered negligible. To disregard a factor of ten times seems to indicate that they were not working with fine units. It is significant that they found no change in the light sense in early glaucoma—only in advanced cases. Although their work covered a great deal of ground and included many excellent features, it appears that all factors were not adequately controlled. We believe, moreover, that Tscherning glasses are not so well adapted for use with the ordinary patient in the clinic as is a machine by which the light can be made brighter or dimmer at will. With Tscherning glasses, the light is not continuously variable, but is cut down by cetrain definite amounts by the application of one of the glasses. The patient is required to state when he is first able to see the test object through the glass. If he happens to be looking somewhere else at that moment he will probably give his report somewhat late. There is no way of estimating the lag involved in making a judgment. Tscherning glasses require constant attention and trained observers.

Rutgers' work [7] also was concerned with much the same problem as ours—the course of dark adaptation. His pathologic cases, however, included retinitis pigmentosa, optic neuritis and choked disk, but no glaucoma. He was particularly interested in the relation between the area of the retina affected by the disease and the part of the adaptation curve that was above normal. He assumed that the level of the first readings depends on the integrity of the central field, and that of later readings on the peripheral field. His results tended to confirm this assumption, but not definitely enough, he believed, to be of value in differential diagnosis. He also studied the effect on the rate of adaptation of contracted and dilated pupils. He did not, however, measure and correct them for variations in size.

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6. Möller (footnote 5, second reference).

7. Rutgers, G. E.: Zur Dunkelanpassung, Klin. Monatsbl. f. Augenh. **71**:449, 1923; Die Dunkeladaptation bei einigen Krankheiten, ibid., p. 589.

Merrill and Oaks [8] attempted to measure the light sense in ordinary daylight by means of Percival's rotating disks. Their light difference test was done with white disks with black sectors which, when rotated, produced the effect of gray circles on a white background, the least amount of gray perceptible on a white background being the light difference. For the light minimum test they used black disks with white sectors, producing the effect of gray circles on a black background. The least amount of gray which could be seen against the black background they called the light minimum. We would point out, however, that this is merely light difference at a lower intensity, since there is no black pigment or dye which does not reflect some of the incident light. Since the test was done in bright daylight, there must have been considerable light reflected from the black. These investigators did not control the intensity of the light on the disks, the pre-exposure or the size of the pupils. They did not work with small differences, having only five possible readings. Their work does not seem to us, therefore, of a high degree of accuracy.

Krüger [9] attempted to measure the light difference by Maxwell disks, but did not have adequate control of the physical factors involved.

Flügel [10] developed a rough test for getting threshold readings of a square of light in a dark room. He did not standardize the physical factors.

Jones,[11] following Flügel's work, with much better developed apparatus, tested the light minimum and visual acuity of twelve aviators at low illumination. The apparatus he used was so arranged that when the patient fixed on a red light, the light sense of the peripheral retina, as well as that near the macula, could be tested. The light intensity was controlled by changing the resistance. He found a wide variation in the light sense among his twelve observers, some of which can probably be explained by the fact that he did not measure the pupils. He found little difference in the light sense between an area 10 degrees around the macula and the peripheral retina out as far as 60 degrees. In his experiment, there did not seem to be any correlation between the light sense and the visual acuity at low light intensities.

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8. Merrill, H. G.: The Light Sense, Eye, Ear, Nose & Throat Monthly **5**:268, 1926. Merrill, H. G., and Oaks, L. W.: The Light-Sense: Its Importance and Tests, Am. J. Ophth. **9**:38, 1926.

9. Krüger, Hans: Ueber die Unterschiedsempfindlichkeit für Beleuchtungseindrücke, Ztschr. f. Psychol. **96**:96, 1924-1925.

10. Flügel, J. C.: Minor Study of Nyctopsis, Brit. J. Psychol., 1920-1921, vol. 2.

11. Jones, L. Wynn: A Method of Measuring Nyctopsis, Brit. J. Psychol. **2**:299, 1920-1921.

Others besides Jones who studied the light sense in the peripheral retina are Mitra,[12] Lo Cascio [13] and Kleczkowski.[14]

Still another problem dealing with light sense, but not related to our work, is the determination of the interrelations of the time of the rise of sensation, the time of exposure and the area of retina stimulated. Piéron,[15] Vogelsang,[16] Kronenberger [17] and Kovács [18] were among the investigators of this problem.

Müller [19] studied light minimum with monocular and binocular fixation.

As an aid to studies on dark adaptation, Drescher and Trendelenburg [20] described an apparatus for obtaining a uniform light adaptation over the entire retina for standardized pre-exposure.

A general criticism which might be made is that practically all investigators of the light sense have worked with normal observers or with cases of well marked diseased conditions, such as retinitis pigmentosa, choked disk, optic atrophy, advanced glaucoma, etc. In such conditions, one can be sure at the outset that the light sense is disturbed, and it has seemed to us that in such conditions a study of the light sense is not of real value, since the best practical index of the progress of these diseases is found in relatively more simple procedures, such as ophthalmoscopic examination and studies of the visual acuity and visual fields. We believe that a more valuable field for light sense studies lies in those

12. Mitra, S. C.: Versuche zur Bestimmung der quantitativen Verhältnisse monokularer und binokularer Lichtempfindungen, mit einer kurzen Einleitung über die Hauptprobleme des indirecktен Sehens, Arch. f. d. ges. Psychol. **55**:1, 1926.

13. Lo Cascio: Il senso luminoso nelle parti periferiche della retina umana, Ann. di ottal. e clin. ocul. **52**:386, 1924.

14. Kleczkowski, T. R.: Die Physiologie und Pathologie der Dunkeladaptation des Auges auf Grund der bisherigen and eigenen Untersuchungen, Arch. f. Augenh. **88**:253, 1921.

15. Piéron, Henri: La loi de l'excitation lumineuse chez Mya arenaria (relation entre l'ntensité et la durée des excitations (luminaires), Mém. Soc. de biol. **93**:1235, 1925.

16. Vogelsang, K.: Der Einfluss der Dunkeladaptation auf den zeitlichen Verlauf der Gesichtsempfindung bei Verwendung farbiger Prüflichter, Arch. f. d. ges. Physiol. **203**:1, 1924.

17. Kronenberger, P.: Empfindungszeit und Empfindungsdauer des hell-und dunkeladeptierten Sehorganes bei Minimalreizung mit farbigen Prüflichterns, Arch. f. d. ges. Physiol. **210**:355, 1925.

18. Kovács, Andreas: Ueber den Einfluss der Dunkeladaptation auf die Empfindungszeit und den zeitlichen Verlauf der Gesichtsempfindung, Ztschr. f. Sinnesphysiol. **54**:161, 1922.

19. Müller, Erwin: Die monokulare und binokulare Reizschwelle des dunkeladaptierten Auges, Arch. f. d. ges. Physiol. **193**:29, 1921-1923.

20. Drescher, K., and Trendelenburg, W.: Eine Lichtflache zur Normierung der Helladaptation, Klin. Monatsbl. f. Augenh. **76**:776, 1926.

cases in which ordinary clinical tests fail to give sufficiently definite information.

PROCEDURE

In the earlier work on the light sense done at the Massachusetts Eye and Ear Infirmary, it was found necessary to fix the observer's pupils with pilocarpine, since obviously the amount of light entering the eye depends on the size of the pupil as well as on the brightness of the object viewed. This procedure was followed in the present series.

After the pupils are fixed, the patient enters the dark room and is seated in a chair so placed that the light source is 1 meter behind his eye, and the screen on which the light is projected for the light minimum test is 4 meters in front of the eye.

A screen, 3 feet (91.44 cm.) square, with two hinged side extensions, each 3 by 2½ feet (91.44 by 76.20 cm.), is placed in front of the patient 70 cm. from his eye. This screen is covered with fine weave white canvas painted with a matt surface white paint. On the bottom is tacked a white cloth which is placed in the patient's lap. The whole arrangement covers practically the entire visual field of the patient. The illumination on this screen is provided by two sets of lamps on adjustable stands on each side of the patient and sufficiently far behind him so that they are not in the field of view, and so arranged that the entire screen is evenly illuminated. These lamps are run on the ordinary house current, which we believe is steady enough for this purpose. The intensity designated as "low" is provided by two 25 watt bulbs; "high" by two 100 watt bulbs. The light from the bulbs is filtered through diffusing screens made of fine weave white linen tracing cloth which gives an even smooth light on the screen. No image of the filament is seen. The brightness of the screen, measured by a Macbeth illuminometer, when lighted by the low lights, is about 2.6 millilamberts; when lighted by the high lights it is approximately 18 millilamberts.

The patient is allowed to become adapted to the lower of these two brightnesses for two minutes. With the screen still illuminated, the light spot[21] is projected on to the screen at about the level of the patient's eyes, and readings are taken of the brightness of the spot, which is required in order that it shall be perceived as different from the background. The procedure is then repeated for the higher brightness. Preliminary investigation showed that to these high intensities adaptation was complete in one or two minutes, and that no change in the readings was discoverable after five, ten or fifteen minutes. Covering one eye while readings are being taken with the other constitutes adaptation to darkness for that eye, and the same time of two minutes must be allowed to elapse after the second eye is uncovered and before its readings are taken.

The regular light minimum test is then done, the time spent with the bright lights on the screen (and enough more to make up ten minutes) being used as pre-exposure time. After these tests, the patient's pupils are measured with the slit lamp and the results corrected for the area of the pupil.

The light difference test was given to eighty-nine patients of our regular glaucoma series (cases in which glaucoma is suspected, usually cases in which one eye has primary glaucoma and the second eye is still

21. The mechanism that produces this light spot was described at length in a previous article (Waite, Derby and Kirk: Tr. Ophth. Soc. **45**:301, 1925). Briefly, it is a projection lantern using a system of photometric wedges to cut down the intensity of light.

clinically normal). Besides these cases in which glaucoma was suspected, we tested also, for comparison, twenty-nine normal persons (fifty-eight eyes) and thirty-eight patients (forty-five eyes) with established glaucoma. So far as possible, normal patients were chosen who were approximately the same ages (from 50 to 70) as the average patient with glaucoma. At the conclusion of the series, we had accumulated records of 130 patients (204 eyes) who had taken the light difference test. Twenty patients (twenty-eight eyes) had taken the test more than once. They had all taken the light minimum test.

Our first step in analyzing the results from these patients was a careful study of the clinical observations as recorded on the examination sheets that are filled out for all patients in whom we are investigating the light sense, and on their clinical record cards. The patients were classified into six groups:

1. Normal—no suspicion of glaucoma.
2. Normal as to history, fundus examination, fields and tension, but with primary glaucoma in the other eye. These patients might be considered more likely to develop glaucoma than those in group 1, but so far no clinical signs of the disease have been discovered.
3. Some slight evidence of future glaucoma. Perhaps the disk was somewhat suggestive, but the tension and fields were normal or there might have been a slight increased tension with everything else normal.
4. Early glaucoma without field changes. Increased tension and a condition of the disk suggestive of glaucoma.
5. Early glaucoma with slight field changes.
6. Advanced glaucoma with large field changes.

Groups 1 and 2 were considered normal; in group 3 glaucoma was suspected, and in groups 4, 5 and 6 glaucoma was present.

The results of the light minimum test were represented by curves on a diagram such as the one shown in chart 1.[22] The numbers along the bottom of the diagram represent the times at which the readings were taken; the numbers along the left side represent the brightness of the spot when it was at the point at which the patient could just see it. The units of brightness are micromillilamberts (signifying 0.000000001 lambert). Ten micromillilamberts, for example, is about the brightness of a piece of ordinary white paper with an illumination of 0.000012 foot

22. In two earlier articles (Waite, Derby and Kirk: Tr. Ophth. Soc. **45**:301, 1925; Further Studies on the Light Sense in Early Glaucoma, Arch. Ophth. **55**: 574 [Nov.] 1926), the curves representing change in brightness threshold during adaptation to darkness were plotted arithmetically. In later work, we have plotted changes in brightness threshold on a logarithmic basis, because it corresponds more closely to the eye's sensitivity to brightness and does not underestimate variations from the normal in the lower range of brightnesses and overestimate those in higher ranges, as does the arithmetical plot. A logarithmic plot, also, enables us to represent the entire course of dark adaptation on a single graph.

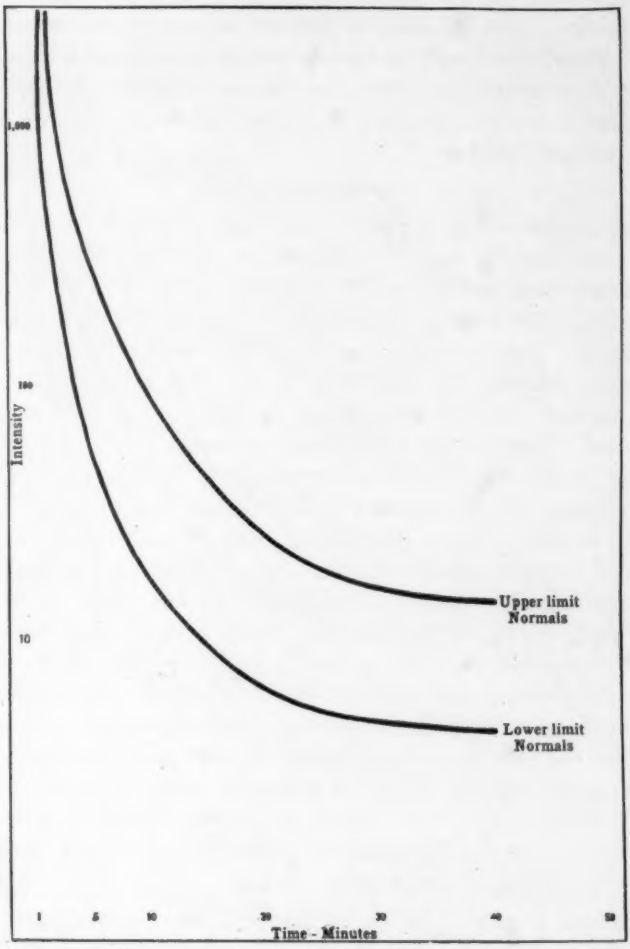

Chart 1.—Light minimum test curves, showing the course of dark adaptation for normal eyes. The numbers along the bottom of the diagram represent the times at which the readings were taken; the number along the left hand side represent the brightness of the spot when it was at the point at which the patient could just see it. The units of brightness are micromillilamberts (signifying 0.000000001 lambert). Ten micromillilamberts, for example, is about the brightness of a piece of ordinary white paper with an illumination of 0.000013 foot candles on it. This diagram shows the limits of normal cases; for example, after forty minutes in the dark a normal patient distinguishes the spot when its brightness is between 5 and 15 micromillilamberts.

candles on it. This diagram shows the limits of normal cases; for example, after forty minutes in the dark, a normal patient distinguishes the spot when its brightness is between 5 and 15 micromillilamberts. Chart 2 shows the types of curves usually associated with glaucoma: *A*, when dark adaptation takes place slowly, although it finally reaches normal limits, and *B*, when dark adaptation takes place slowly and never reaches normal limits.

LIGHT DIFFERENCE

When the eyes of each patient were classified with respect to clinical observations and the light minimum test, a comparison was made of these observations with a view to determining the value of the light difference test. The results of the light difference test are represented by percentages. When a spot of light is projected on a bright screen, it is necessary to increase the brightness of the spot a certain amount over that of the screen before the spot can be perceived as different from the background. The amount of increase necessary for a difference to be perceived varies with the brightness of the background, and in the ordinary ranges of illumination is a constant percentage of the background. In the range of illumination that we have used, the smallest perceptible increase in brightness has been demonstrated by many investigators [23] to be approximately 2 per cent. In our work, we found this percentage to average 2 per cent for clinical group 1 (normal); 2.8 per cent for group 2; 3 per cent for group 3; 3.5 per cent for group 4; 6.1 per cent for group 5, and 6.5 per cent for group 6. Thus, the averages showed differences between the various clinical groups; for example, the average normal patient could perceive a difference of 2 per cent, whereas the average patient with advanced glaucoma could not perceive a difference of less than 6 per cent. We found, however, that there was a wide variation in readings of light difference percentages in each clinical group and much overlapping between the groups.

There was a wide variation of readings, also for one person over a period of time. Twenty patients (twenty-eight eyes) took the light difference test more than once, at an average interval of two months. We found poor reproducibility; i.e., the two readings by the same patient did not agree. This suggests that there is some intrinsic difficulty in the test itself. Those who have tried the test have found it much harder to judge when the spot is seen against the bright background than in the regular light minimum test in which the surroundings are dark. This difficulty seems to be due mostly to after-images and fatigue and it is complicated by the length of time the observer takes in deciding whether or not the spot is present. We have tried to think of some way of mak-

23. Hecht (footnote 2). Aubert, König and Brodhun and Blanchard (footnote 4, first, second and third references).

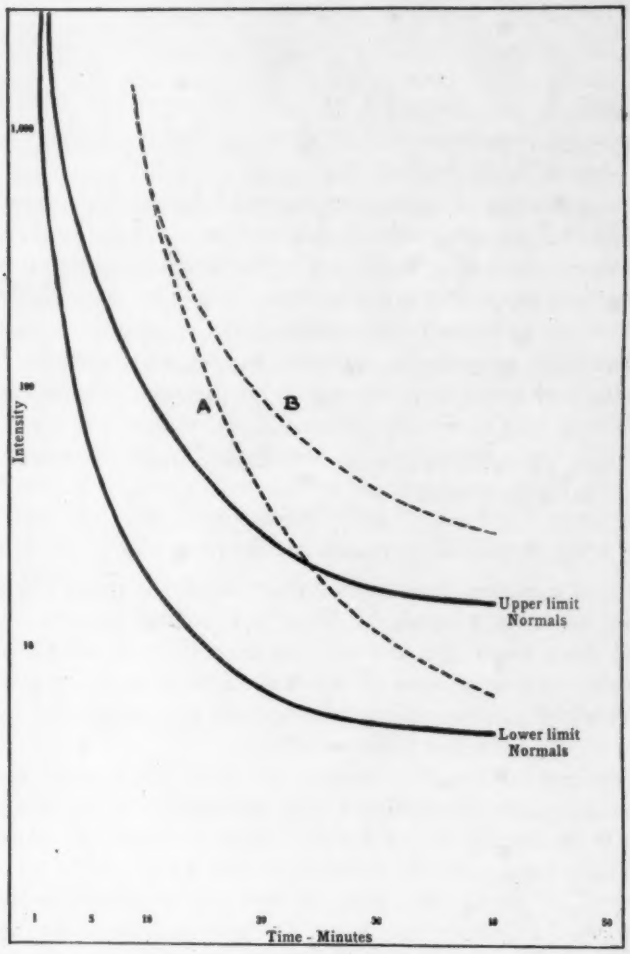

Chart 2.—Light minimum test curves, showing the course of dark adaptation of the types usually found in glaucoma. *A*, dark adaptation takes place slowly (i. e., after ten minutes the patient requires 600 micromillilamberts' brightness of the spot in order to see it, while the normal patient usually sees the spot when its brightness is about 50 micromillilamberts), but finally reaches normal limits; *B*, dark adaptation takes place slowly and never reaches normal limits.

ing the judgment more exact and easier, but without success. Because of these difficulties and of the great variation in the results, we have come to the conclusion that tests of the light difference are too variable and inaccurate to be of clinical value.

SUMMARY

1. A test of the perception of light difference has been made in normal persons, in patients with preglaucoma and in patients with established glaucoma in its various stages.

2. The smallest perceptible increase in brightness that ranged, on the average, from 2 per cent in the normal person to 6.5 per cent in patients with advanced glaucoma; however, a wide variation in these percentages was found among patients of the same clinical group, and the results in a given case were not reproducible.

3. This wide variation is probably the result of difficulty in judgment on the part of the observer, due to the presence of after-images and fatigue.

4. A test of light difference at ordinary illuminations is not significant in early glaucoma.

LIGHT MINIMUM TEST

The light minimum test, on the other hand, has proved increasingly significant in early glaucoma. Since our technic became sufficiently perfected three years ago, the test has been done on all patients with preglaucoma who have come to the clinic as well as on an increasingly large number of patients referred to us from private practice by various members of the infirmary staff and others.

The patients with preglaucoma are tested regularly every six months until the diagnosis of glaucoma can be established by the ordinary methods of tonometry and perimetry. A large number of patients have had the light minimum test more than once—many five, six or seven times. It is in these cases that we find the most interesting results. Some of the patients whose cases we have followed have developed clinical glaucoma, and most of these had shown a rise in the light minimum curve while under observation. Many pateints whose light minimum curve has been getting higher have not yet shown positive clinical signs of glaucoma; however, it is these cases that are watched most carefully in our follow-up of glaucoma. On the basis of our present experience with the light minimum test, glaucoma would seem to be more likely to develop in these patients than in those whose light minimum curve stays consistently at a low level. In normal cases and in cases of established glaucoma, we have found that the light minimum test agrees satisfactorily, on the whole, with the clinical observations,

although there are exceptions. In cases of incipient glaucoma, we believe that a rise in the light minimum curve is often the earliest sign.

COMMENT

We believe that a test of the light sense is valuable in ruling out or recognizing early glaucoma. Is there any way in which this may be made available for the profession in general? The apparatus which we are using was specially built to afford a wide range of intensity while keeping the color value the same, and to insure the greatest possible degree of accuracy in controlling and measuring the exact amount of light delivered. While admirably designed for use in a hospital such as ours, where large numbers of cases are available, it is bulky, not transportable and probably unnecessarily complicated for ordinary clinical work. While we do not think that simple forms of apparatus like Percival's disks are of any value for showing up such small differences in the light sense as occur in early glaucoma and that the same applies to the tests made with Tscherning glasses as now performed, we suggest that there are simple forms of photometers which, when used with accurate control of such factors as pre-exposure of the patient's eyes and fixation and measurement of his pupils, will give sufficiently accurate results to be of clinical significance. We hope to report on this question later.

A TUMOR (CAVERNOUS ANGIOMA) OF THE ORBIT CAUSING PRESSURE SYMPTOMS ON THE EYEBALL*

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Tumors of the orbit sometimes press on the eyeball, causing indentation of the sclera with curious ophthalmoscopic symptoms.

REPORT OF A CASE

Mr. L. D. O., aged 36, was referred to me on Sept. 29, 1927, through the courtesy of Dr. D. T. Bishop of Brooklyn, with the history that there had been a slowly developing protrusion of the left eye for some years. The vision in the left eye with a +2.5 diopter lens was 20/20; the right eye was myopic and vision was corrected to 20/20 by a —1.25 —0.75 cylinder 90 degree lens. The bulging of the left eye was 21 degrees (exophthalmometer) as compared with 16 of the right. The palpebral fissure of the left eye was 6.5 mm., while that of the right was 5 mm. Palpation within the orbital margin gave negative results. The motility of the eye was normal, except for a paresis of the superior rectus. The pupil, visual field and blind spot were normal. The fundus presented the following interesting condition: there was a mottling and blurring of the region about the nerve head with dilatation of the vessels. This blurring extended over to include the macular region and contained a series of horizontal lines. These lines were easily observed and extended from the disk to the macula, suggesting folds in the retina. This area was best seen with a +3.5 to +5 diopter lens. This prominence shaded off gradually without any ophthalmoscopic signs.

The combination of this ophthalmoscopic picture of a circumscribed prominence of the eyeground and exophthalmos suggested an orbital tumor closely connected with the eyeball. Direct involvement of the sclera and choroid could be excluded by the absence of choroidal changes and optic neuritis. On the other hand, this ophthalmoscopic condition made it uncertain whether the tumor, if one were found, could be removed without injuring the eyeball.

The roentgen report showed a clouded left orbit with obscuration of bony details. The sinuses were normal, and the optic canal was normal.

A Kroenlein operation was performed on Feb. 2, 1928, the circular saw driven by the surgical hand motor being used and the usual technic being followed. A bluish, smooth tumor was readily detected behind the eyeball, extending back and up over the optic nerve. It was encapsulated and was easily shelled out. Recovery was prompt, and the cosmetic result was satisfactory.

The tumor measured 20 by 12 mm. after alcohol fixation, and was egg-shaped.

The microscopic examination revealed the usual signs of a cavernous angioma.

The haze and the folds in the eyeground had disappeared on February 17. One week later, the exophthalmometer measured 15 degrees in each eye. The refraction at first remained unchanged, then gradually weaker plus glasses were

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* Read before the Section of Ophthalmology, New York Academy of Medicine, Jan. 21, 1929.

accepted, and at the last examination, Nov. 30, 1928, 20/20 vision was obtained with a +2 diopter lens. The fundus was normal except for some faint lines along the superior temporal vessels.

In this connection, it is interesting to note that Dr. Bishop when he first saw this patient in 1920 found the refraction to be −0.25 +0.50 cylinder 120 degrees. Three years later, the patient complained of metamorphopsia and the refraction had changed so that a +2.25 diopter lens was required. In July, 1927, with a +3.5 diopter lens, the vision was 6/6, and an exophthalmos was beginning to develop.

Dimmer [1] has spoken of a number of changes in the eyeball which result from the pressure of tumors, the first consisting in a disturbance of the nutrition of the retina in the macular region. Haab has also spoken of this and has observed the appearance of black points and later a yellowish-red spot in the macula. In another case, there was absence of the foveal reflex, and there were oblique lines at the margins of the macula. This is presumably due to pressure on the eyeball from behind which causes a flattening and indentation of the posterior pole. The relief of the inner surface is changed, and the innermost surface becomes uneven.

Dimmer,[2] in speaking of scleral indentation, said that the choroid is raised just as the retina is raised by a choroidal tumor. Leber and Hirschberg were the first to speak of indentation of all the ocular membranes from pressure of an orbital tumor. Haab drew attention to the similarity of this appearance to that of an intra-ocular tumor, and described the condition as a particular form of phantom tumor of the eye.

Loehlein [3] described the formation of folds in the eyeground from the pressure of a mucocele of the frontal sinus. These differ from the folds in the retina described in various conditions by Vogt.[4] They occurred at the posterior pole, extending from the macula in all directions for a distance of 4 papilla diameters. They were most marked in the vicinity of the macula and optic disk. They are generally horizontal, describing a slightly concave arch, increasing upward. After the operation the disturbance in vision was lost, but the folds in the retina remained. Loehlein expressed the belief that the changes had been made permanent, as they had existed for more than one and one-half years, and that they were caused by folds in the inner layers of the retina.

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1. Dimmer: Der Augenspiegel, ed. 3, Leipzig, F. Deuticke, 1921, p. 456.
2. Dimmer (footnote 1, p. 622).
3. Loehlein: Ztschr. f. Augenh. **64**:164, 1928.
4. Vogt: Arch. f. Ophth. **99**:330, 1919.

OCULAR ANAPHYLAXIS *

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The earliest observation published on protein anaphylaxis was reported by Magendie [1] in 1839. He described the sudden death of dogs that had been given repeated injections with egg albumin.

In 1894, Flexner [2] gave a clear statement of the fundamental phenomenon involved, that is, he showed "that animals that had withstood one dose of dog serum would succumb to a second dose given after a lapse of some days or weeks; even when this dose was sublethal for a control animal." This phenomenon is now known as active sensitization.

The first observations that had any relation to ocular conditions were reported by Uhlenhuth [3] in 1903, in which he showed that animals could be sensitized and immunized with lens protein, and that this hypersensitivity was tissue specific and not species specific. In the same year, Arthus [4] showed that cutaneous lesions resulted at the former site of injection in rabbits repeatedly injected with horse serum.

It is on these facts that subsequent researches dealing with ocular anaphylaxis are based.

The first work that had a direct bearing on ocular anaphylaxis was reported by Schoenberg [5] in 1914. In his experiments, he was able to produce artificial interstitial keratitis and iritis by first sensitizing a rabbit with blood serum from a patient with interstitial keratitis; ten days after a second injection, he injected the serum into the parenchyma of the cornea.

In 1916, Elschnig [6] demonstrated that uveal pigment has antigenic properties as shown by the complement fixation, and advanced the theory that sympathetic ophthalmia is an allergic reaction of the uveal tissues in the sympathizing eye in a patient hypersensitive to uveal pig-

* Submitted for publication, February, 1929.
* Read before the Sioux Valley Eye and Ear Academy, Feb. 22, 1929.

1. Magendie: Vorlesungen über das Blut, 1839, in Ehrlich: Gesammelte Arbeiten, tr. by John Wiley & Sons, New York, 1906, p. 332.

2. Flexner: Med. News **65**:116, 1894.

3. Uhlenhuth: Zur Lehre von der Unterscheidung verschiedener Eweissarten mit Helfe spezifischer Sera, Koch's Festschr., 1903.

4. Arthus: Injections répété de serum de cheval chez le lapin, Compt rend. Soc. de biol. **4**:817, 1903.

5. Schoenberg, M. J.: A Contribution to the Experimental Study of Ocular Anaphylaxis, New York State J. Med. **14**:493, 1914.

6. Elschnig, A.: Studien zur sympathischen Ophthalime: II. Die Antigene Wirking des Augenpigmente, Arch. Ophth. **76**:590, 1910.

ment. He also brought out the fact that uveal pigment was tissue specific, and not species specific. The following year, Alan Woods [7] confirmed and elaborated on the observations of Elschnig.

Igersheimer [8] claimed that in children with hereditary syphilis spirochetes are present in the cornea which sensitize it to the condition. Later, if spirochetes become active in some other part of the body and liberate their toxins in the blood, they cause an anaphylactic reaction on the hypersensitive cornea. This condition is called parenchymatous keratitis.

In the same year, von Szily [9] advanced the theory that phlyctenules, which he considered tuberculous, asthma and spring catarrha, are anaphylactic in origin. Since that time, a number of authors have confirmed these observations, some of whom, however, have given a different interpretation to their observations.

In 1920, Aubineau [10] suggested the theory that recurrent intra-ocular hemorrhages of adolescence are probably due to anaphylactic changes in hemophilia.

In 1922, at the International Congress of Ophthalmology, Verhoeff and Lemoine [11] advanced the theory that intra-ocular inflammation following rupture of the capsule of the lens, spontaneous, operative or traumatic, in which no active infection existed, is an anaphylactic reaction to liberated lens substance in a patient hypersensitive to lens protein. Twelve cases were reported in which intra-ocular inflammation was apparently due to lens substance, and all twelve patients gave a positive reaction to lens protein. The microscopic examination of five eyes from these patients showed a characteristic histologic picture in each. About 8 per cent of all adults were found to be hypersensitive to lens protein.

In the same year, Verhoeff and Lemoine [12] reported the case of a patient, markedly hypersensitive to lens protein, who was desensitized, as demonstrated by a negative reaction to an intradermal test, and successfully operated on for immature cataract by discission and linear extraction.

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7. Woods, A. C.: Ocular Anaphylaxis: III. The Rôle of Uveal Pigment, Arch. Ophth. **46**:283 (July) 1917.

8. Igersheimer, J.: Destiny of Patients with Parenchymatous Keratitis of Hereditary Syphilis, Thirty-Ninth Ophthalmic Congress, Heidelberg, p. 283.

9. Von Szily, A.: Anaphylaxis in Ophthalmology, Clin. Opht. **21**:251, 1917.

10. Aubineau: Anaphylaxis Provoked by Treatment of Intraocular Hemorrhages in Adolescents, Arch. Ophth. **31**:372, 1920.

11. Verhoeff, F. H., and Lemoine, A. N.: Endophthalmitis Phacoanaplylactica, Tr. Internat. Cong. Ophth., 1922, p. 234.

12. Verhoeff, F. H., and Lemoine, A. N.: Hypersensitivity to Lens Protein: Patient Desensitized and Successfully Operated on for Immature Cataract, Am. J. Ophth. **5**:700, 1922.

The following year, Lemoine and MacDonald [13] reported 168 unselected cases in which tests were conducted for hypersensitivity to lens protein in which the capsule of the lens had been ruptured by either operation or trauma. Fourteen of the patients gave positive reactions of varying degrees. In seven of the fourteen patients, a fair amount of free cortical substance remained, and intra-ocular inflammation developed. The inflammation varied with the amount of cortical matter left and the degree of hypersensitivity of the patient. In one case, the microscopic observations were identical with those reported by Verhoeff and Lemoine. Verhoeff and Lemoine also reported two cases of patients markedly hypersensitive to lens protein, who were desensitized as demonstrated by a negative reaction to an intradermal test and successfully operated on for immature cataract by discission and linear extraction. An unsuccessful effort was made to desensitize a patient with severe endophthalmitis phaco-anaphylactica who was markedly hypersensitive to lens protein. In none of the patients giving a negative reaction to lens protein did an intra-ocular inflammation develop.

In an excellent article, Alan Woods [14] reported a number of cases of conjunctivitis which recurred in the summer; all of the patients gave positive reactions to one or more pollens. He also reported the cases of nineteen patients with vernal catarrh, all of whom gave positive reactions to some of the pollens. Thirteen were improved, and one was cured by treatment with pollens. He also reported the case of a patient with keratitis, who proved to be hypersensitive to corn. This patient was desensitized with corn pollen, and the disease subsided, with good end-results.

In 1925, Gifford and Steinberg [15] largely confirmed the results obtained by Verhoeff and Lemoine and those by Lemoine and MacDonald. However, they concluded from their experimentation on animals that cataractous lens in the anterior chamber was more toxic than normal lens substance.

In 1925, at the American Academy of Ophthalmology and Oto-Laryngology,[16] I reported the cases of six patients with vernal catarrh, five of whom were sensitive to pollens or foods. Six patients who had recurrent conjunctivitis each summer were sensitive to one or more pol-

13. Lemoine, A. N., and MacDonald, A. E.: Observation on Phacoanaphylactic Endophthalmitis, Tr. Am. Ophth. Soc., 1923; Phacoanaphylactic Endophthalmitis, Arch. Ophth. **53**:101 (March) 1924.

14. Woods, A. C.: The Application of Immunology to Ophthalmology, Arch. Ophth. **53**:321 (July) 1924.

15. Gifford, S. R., and Steinberg, A. A.: Allergic and Toxic Properties of Lens Protein, J. A. M. A. **85**:351 (Aug. 1) 1925.

16. Lemoine, A. N.: Allergies in Ophthalmology, Tr. Am. Acad. Ophth. & Oto-Laryng., 1925, p. 198.

lens. Also, the cases of four patients with trachoma were reported who had recurrences each summer and were hypersensitive to pollens. The cases of five patients with phlyctenular keratitis were reported, four of whom gave positive von Pirquet reactions, and one was sensitive to strawberries. One patient had severe conjunctivitis due to face powder to which she was hypersensitive. Two cases were also reported of patients with recurrent dendritic ulcers, one of whom was hypersensitive to chocolate, and one to pollens. In the discussion of that paper, the late Lee Francis reported a case of herpes of the cornea of several weeks' duration. The patient was found to be sensitive to chocolate and corn; the withdrawal of these foods from her diet resulted in prompt healing of the herpes with a recurrence when they were again used. The following year, she was able to eat a moderate amount of both chocolate and corn without any recurrence of herpes.

In 1927, I [17] reported the case of a patient who developed severe conjunctivitis and dermatitis from butyn. This patient had one relapse when butyn was again used.

Since that time, I have had one patient, a man, who frequently had attacks of diplopia associated with severe vertigo and scintilating scotoma. These attacks were not followed by headaches. At the time of examination, his eyes were normal, with a normal muscle balance. He was examined by an internist, who obtained negative observations. On going over his history, I concluded that he must be sensitive to cheese. My suspicion was confirmed. Cheese was removed from his diet, and he has never had another similar attack, except after eating this food. This patient is able to use milk in moderation without any symptoms.

In the past two years, I have seen seven new cases of vernal catarrh; all the patients were hypersensitive to one or more pollens, and two were hypersensitive to one or more articles of food. Four of the cases occurred in children. I saw one of the children in the spring of 1928, after the onset of the illness. This case was unusually severe; a large gray fold was present all around both corneas, which covered about half of the total area of the cornea. This patient was sensitive only to spring pollens, and the condition cleared up about June 15. I saw another of the children in the summer of 1926; in 1927 she was immunized to the pollens to which she was hypersensitive, and that summer was practically free from the disease. In the summer of 1928 she had a recurrence, which was not so severe as those of former years. This patient had a hypothyroid condition; she was given doses of thyroid parathyroid and calcium. Within a few days, she was free from all symptoms. The

17. Lemoine, A. N.: Conjunctivitis and Dermatitis Due to Butyn, Am. J. Ophth. **10**:125 (Feb.) 1927.

three adults were 22, 26 and 30 years of age. In 1926, these three patients were immunized to the pollens to which they were hypersensitive, and all were well that year; all three had recurrences the following summer, however, although not so severe as on previous occasions. Two of them had hypothyroid conditions. They were given thyroid-parathyroid with calcium, and both improved following the treatment. The other patient was given parathyroid with calcium, and the condition immediately improved.

In the same period, I saw eight cases of phlyctenular keratitis in children; all of whom gave positive von Pirquet reactions; all made a complete recovery after the treatment with tuberculin.

COMMENT

From the foregoing review, it seems certain that allergies play a definite rôle in ophthalmology; however, after studying and observing some of these cases over a period of six years, I have come to the conclusion that in most cases allergy is not the underlying cause, but merely a manifestation of a disturbed endocrine balance with a resulting disturbance of the metabolic and biochemical function.

The great majority of cases of vernal catarrh occur in children. The few cases I have seen nearly all subsided when the patient reached puberty, and most of the symptoms entirely disappeared. At puberty, the gonads assume a new function, and the thymus gradually loses its function. At this time in life, goiter develops in many patients; in fact, at this time in life a complete readjustment of the whole process of internal secretions takes place. Two of the three patients I have seen with vernal catarrh who were past puberty had hypothyroidism. One of them had had vernal catarrh since early childhood; the other developed it at puberty. Both recovered following the administration of thyroid-parathyroid with calcium. The condition of the third improved after treatment with parathyroid and calcium.

One of the patients with dendritic keratitis had just passed the menopause. The patient who developed hypersensitiveness to butyn had also just passed the menopause. The case reported by the late Lee Francis was that of a patient, aged 18, who apparently became immune spontaneously to chocolate and corn in a year.

Lagrange [18] reported the case of a patient in whom vernal conjunctivitis developed at the age of 30, following the removal of the ovaries; the conditions cleared up after the administration of thyroid and ovarian substance. Lagrange [19] also reported the case of a man, aged 65, in

18. Lagrange, H.: Conjunctivitis of Anaphylactic Origin, Presse méd. **31**:380, 1923.

19. Lagrange, H.: Anaphylactic Origin of Conjunctivitis, Presse méd. **31**:112, 1923.

whom a severe case of conjunctivitis developed as a result of handling wood. He was found to be exceedingly sensitive to oak pulp. This patient had diabetes.

Unfortunately, there is no accurate means of determining the function of most of the glands of internal secretion, and knowledge of normal biochemical disturbances is so limited that in many cases it is impossible to come to a definite conclusion.

A careful history is essential in those cases, followed by a complete medical examination, including a basal metabolism test. If no abnormal condition is found, one has to depend on the history and the general characteristics of the patients to determine if any of the endocrine glands have a disturbed function. From my experience, it seems that the thyroid, the parathyroid and the gonads are the glands most frequently disturbed in patients who develop hypersensitivity to proteins.

On account of its interrelation with the thyroid and the gonads, the pituitary gland, of necessity, must also be disturbed; but, again, the diagnosis of dysfunction of the pituitary gland is difficult, and so far I have not had a case that I felt justified the therapeutic test.

TREATMENT

The treatment for ocular anaphylaxis has as its object correction of the endocrine disturbance. The antigen causing the allergy should be identified and removed from the patient if possible. If it is impossible to remove the antigen from the patient, then the patient should be immunized to the antigen in question; apparently, this immunization must be repeated about every two years.

CONCLUSION

Allergy plays an important rôle in many ophthalmic clinical entities.

The great majority of patients developing the allergic phenomena have endocrine disturbances.

Many patients can be cured of these allergic phenomena by glandular therapy, most commonly of the thyroid, parathyroid and gonads.

AN OBSTINATE CASE OF CHRONIC INFECTION OF THE CONJUNCTIVAE FROM BACILLUS XEROSIS*

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In the summer of 1911, in New York, Mr. S. applied to me for help. Years of suffering from intolerable conjunctivitis, which resisted the best therapy, brought him to me in the absence of my senior. I convinced myself that all the sovereign remedies were useless, and certainly did not ease but, rather, aggravated the pain. His justified desire to seek transatlantic help incited me to make the utmost effort to cure him. Smears of the conjunctiva were taken that showed an enormous number of xerosis bacilli and pus cells, but nothing else. Cultures showed pure xerosis and that failed to grow on a medium that was even slightly acid. They perished and lost their virulence when grown and submitted to any acid. This was the crux of the extraordinary cure to follow.

I first tried on my own conjunctiva every conceivable acid, mineral and vegetable, inorganic and organic, that I could obtain. The irritation even in weak solutions was too intolerable for words. But when I tried lemon juice, fresh and filtered, I found that I could stand without distress a 1 per cent and even a 2 per cent solution used in an eyecup. I then had my dispenser test numerous substances, and selected all those that showed an acid reaction. The following prescription was given to my patient:

	Gm. or Cc.
℞ Boric acid crystals	4.0
Sodium chloride	2.8
Filtered lemon juice	2.0
Suprarenaline (A + Co.)	2.0
Camphor water	50.0
Distilled water to make	200.0

S: To be used frequently, at least three times daily, in full strength in an eyecup.

I saw Mr. S. repeatedly afterward, and on Jan. 25, 1914, he had enjoyed two and one-half years of complete comfort. His relief was immediate and lasted, to my knowledge, till I left New York, in 1914. A rather significant accident happened. The patient was in Berlin and took my prescription to an apothecary there. This scientific gentleman seemed to have turned up his nose at lemon juice, in his superior wisdom, thinking citric acid was more professional. He used it. Mr. S. suffered agonies as I well believe, having tried it on myself.

I did not go any further in my investigations, but have no doubt that the juices of many West Indian or Southern citrous fruits, as lime, grapefruit, shaddock, bitter orange, sour orange, sweet orange, etc., might act as well.

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THE THERAPEUTIC VALUE OF TUBERCULIN IN OPHTHALMOLOGY*

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The conflicting points of view evidenced by a study of the literature indicate the difficulties that have been encountered by those who since the days of Koch have endeavored with all the energies at their command to arrive at a proper evaluation of tuberculin as a therapeutic agent. The position of the ophthalmologist who contemplates the use of tuberculin is especially difficult. Tuberculin is not generally employed by internists in this country in the treatment for tuberculosis, and its use by the ophthalmologist is likely to be looked on by the family physician with disapproval. There is a prevailing opinion among internists that the diagnosis of ocular tuberculosis must be supported by decided evidence of pulmonary tuberculosis. There is also a skepticism among nonophthalmologists as to the occurrence of ocular tuberculosis, except in rare instances. Rest, hygiene and dietetic measures in the treatment for tuberculosis are not fraught with danger; this is not true of tuberculin. In regard to its use, there is complete unanimity of opinion on at least one point, and that is that when it is improperly used it is capable of doing great harm. Tuberculin is intimately related to the tubercle bacillus and its manifestations in the body. Any one who utilizes it clinically, even though such use unfortunately is largely empiric in the present state of knowledge, must of necessity have as a background for safe and adequate tuberculin therapy a knowledge of the fundamental principles of the disease, tuberculosis, in general. A source of added responsibility for the ophthalmologist in treating patients with ocular tuberculosis arises from the fact that a relatively minute tissue destruction in the eye may imply an interference with or loss of function out of all proportion to that produced by a lesion of similar extent in another organ. This consideration has, I believe, some important implications, to which reference will be made later. Ranke,[1] to whose epoch-making

* Submitted for publication, March 26, 1929.

* Read at the New York Academy of Medicine, Section on Ophthalmology, Feb. 18, 1929, proceedings of which, including discussion on this paper, appear in this issue, p. 754.

1. Ranke, K. E.: Primäraffekt, sekundäre und tertiare Stadien der lungentuberkulose (three sections); I. Die primäre Lungentuberkulose des Menschen, Deutsches Arch. f. klin. Med., 1916, vol. 119; II. Lungenerkrankungen bei generalisierter Tuberkulose und isolierter Phthise, ibid., 1916, vol. 119; III. Die Abgrenzung der Stadien innerhalb des Gesamtgegiestes der menschlichen Tuberkulose, ibid. **129**: 224, 1919; Primäre, sekundäre und tertiare Tuberkulose des Menschen, München. med. Wchnschr. **64**:320, 1917; Die Beteiligung der Lunge an den allergischen Stadien der Tuberkulose, Beitr. z. klin. d. Tuberk. **52**:212, 1922; Bemerkungen zur klinischen Diagnose der Entwicklungsformen der menschlichen Tuberkulose, München. med. Wchnschr. **69**:69, 1922; Ausgewählte Schriften zur Tuberkulosepathologie, Berlin, Julius Springer, 1928. This book contains an excellent critique of Ranke's interpretation of tuberculosis and disease in general.

researches on the pathology of tuberculosis most of the present knowledge is due and whose work Turban,[2] in a comparatively recent article on the classification of tuberculosis, extolled as the greatest contribution to the understanding of tuberculosis since the discovery of the tubercle bacillus by Koch, had the following to say concerning tuberculin: "It is possible by tuberculin to change a relatively beneficient immunity into (what Ranke terms) the specific toxin hypersensitiveness of the immune." According to Ranke, tuberculin in its therapeutic application releases only the spontaneous reactions of the prevailing state of the disease. Its use does not imply the introduction of a protective agent, he stated, but rather the provoking of a disease. Any one who is familiar with the manifold variations in tuberculosis in man will not be surprised that the artificial employment of the same virus, the spontaneous production of which is so incalculable, cannot be reduced to a simple formula. Ranke, however, maintained that the proper therapeutic use of tuberculin is only a matter of time.

A number of studies of the ultimate results and clinical course of cases of ocular tuberculosis extending over a period of years, with reference to the value of tuberculin, have been reported in the literature. Von Hippel's[3] results are based on a study of 243 patients. He reported 75.7 per cent cured, 17.3 per cent improved and 7 per cent failures. Schieck[4] reported sixty-four failures in 116 cases from the Göttinger material in which no specific therapy was undertaken. As the result of a survey extending over twelve years, Bernheimer[5] favored the specific treatment for scrofulous keratitis. During the first six years before specific treatment was instituted, the recurrences amounted to 50 per cent; during the six years after specific treatment was used, recurrence occurred in only 20 per cent of the cases. Hertel,[6] writing in 1914, emphasized the fact that in previous publications the occurrence of spontaneous cure had not been sufficiently considered. He divided his material of seventy cases into three groups: the first group includes that in which no benefit was obtained through the use of tuberculin. It comprised fourteen cases of iridocyclitis, two of keratitis and three of

2. Turban, L.: Zur Klassifikation der chronischen Lungentuberkulose. Ein Vortrag mit Schema, Ztschr. f. Tuberk. **40**:404, 1924.

3. Von Hippel, A.: Ergebnisse der Tuberkulinbehandlung bei der Tuberkulose des Auges, Arch. f. Ohpth. **87**:193, 1914.

4. Schieck, F.: Die abhängigkeit des Verlaufes der tuberkulosen Prozesse am Auge von dem Stadium der Allergie des Gesamtorganismus, Arch. f. Ophth. **105**:257, 1921.

5. Bernheimer, S.: Tuberkulinbehandlung Augenkranker, Klin. Monatsbl. f. Augenh. **16**:579, 1913.

6. Hertel, E.: Ueber den heutigen Stand der Behandlung tuberkuloser Augenerkrankungen, Samml. zwangl. Abhandl. a. d. Geb. d. Augenh. **9**:1, 1914.

chronic retinitis. Among these cases, two eyes had to be enucleated and two resulted in phthisis bulbi despite tuberculin therapy carried on for two months. Löwenstein,[7] in commenting on this report, called attention to the minimum dosage of tuberculin used. In one case of choroiditis in which tuberculin had been repeatedly used, the choroidal exudates increased, and there occurred an eruption of fresh nodules in the iris and deposits on Descemet's membrane. A similar unfavorable progress of the disease was noted in two cases of parenchymatous keratitis and in one case of vitreous hemorrhage. The unfavorable cases are in decided contrast to those of his second group in which marked improvement occurred during treatment with tuberculin, which was maintained during the period of control. In a few cases, recurrences took place but gradually disappeared and did not interfere with an outcome that was ultimately favorable. This group included twenty-five cases, some of which were severe. Some of the patients had previously been subjected to nonspecific treatment with absolutely no benefit. Hertel assigned to a third group those patients whose conditions showed no improvement, although some of them were treated with tuberculin, but who, during the subsequent control period, frequently displayed a surprising tendency to recovery. Hertel did not ascribe this outcome to a delayed therapeutic effect of tuberculin, as the improvement occurred long after the discharge of the patient from active treatment and took place in some cases in which no tuberculin was used. As a result of his study of this material, Hertel estimated that the general average therapeutic effectiveness of tuberculin as contrasted with nonspecific treatment amounts to approximately 20 per cent. As a result of many years' observation, Hess [8] concluded that tuberculin was devoid of influence on the course of ocular tuberculosis, especially that of the uvea. Gilbert,[9] in an article on tuberculosis of the iris in the Graefe-Saemische "Handbuch," displayed a similar pessimism. Meller [10] and Novak,[11] on the contrary, insisted on treatment with tuberculin as imperative in ocular tuberculosis. Their conviction is the result of a survey of the enormous material of the Vienna clinic. Weigelin [12] published the results of a survey of seventy-

7. Löwenstein, Arnold: Die Tuberculose des Auges, Berlin, Urban & Schwarzenberg, 1924.

8. Hess, in Löwenstein: Die Tuberculose des Auges, Berlin, Urban & Schwarzenberg, 1924.

9. Gilbert, W.: Erkrankung des Uvealtraktus, Handb. d. ges. Augenh. Herausg. b. Axenfeld und Elschnig, ed. 2, vol. 5, p. 6

10. Meller, J.: Ueber die Behandlung von Augenkranheiten mit Tuberkulin, Wien. klin. Wchnschr. **35**:193, 1922.

11. Novak, E.: Die spezifische Behandlung der Augentuberkulose durch den praktischen Arzt, Wien. klin. Wchnschr. **35**:193, 1922.

12. Weigelin, S.: Erfahrungen mit der Tuberkulosebehandlung bei Augentuberkulose, Klin. Monatsbl. f. Augenh. **66**:641, 1921.

seven cases of ocular tuberculosis in 1921. The average period of observation in these cases was six and one-half years. He excluded from his statistics cases which had been observed for less than two years. He regarded 41 per cent of his patients as ultimately cured, 34 per cent improved and 24 per cent without benefit. Weiglin [12] applied the term "improved" to those patients whose conditions were quiescent over long periods but in whom recurrence took place despite repeated tuberculin "cures." He reported complete failure in 8 per cent. He emphasized that under certain conditions tuberculin therapy is devoid of effect, but recommended that a trial be made in all cases, as he had never observed any injurious effects from its use. In 1920, Igersheimer [13] subjected the material of David, von Hippel and Schieck to a revision. He compared the material of the Göttinger clinic from 1890 to 1900, that is, during the period preceding the introduction of tuberculin, with the cases occurring during the years from 1903 to 1913, and finally with the material of the years from 1914 to 1915. He concluded that tuberculin was devoid of any beneficial influence, as he encountered many patients who had gone blind despite early improvement under tuberculin therapy. He went so far as to question the specific action of tuberculin and compared it to protein therapy. In 1923, Hartig [14] found, as the result of an examination of the material at Königsberg, a definitely higher percentage of cures with specific as compared with nonspecific treatment. Derby and Carvill,[15] in a paper read before the Ophthalmological Society in 1927, presented a study of the ultimate result of sixty-three cases of anterior ocular tuberculosis followed over a period of years. Their conclusions in regard to tuberculin therapy are that in certain instances it may help to cut short the present attack, that it does not prevent recurrences and that occasionally, especially in inexperienced hands, it may do serious harm. Obviously, they believe tuberculin to be practically devoid of value as a therapeutic agent. In the discussion of the paper, Verhoeff concurred in their opinion. In 1926, Fleisher [16] reported on a series of ninety patients with ocular tuberculosis treated with Tebeprotin and observed over a period of four years. In the subsequent tabulation, I exclude the scrofulous types amounting to forty-one cases, also two cases in which the patients are still under treatment, one case of caries, two cases of periphlebitis and one case of choroiditis. His

13. Igersheimer: Zur Bewertung der spezifischen Tuberkulosetherapie, Ber. d. deutsch. ophth. Gesellsch. **42**:235, 1920.

14. Hartig, O.: Ueber den gegenwartigen Stand der Behandlung der Augentuberkulose, Ztschr. f. Augenh. **50**:79, 1923.

15. Derby, G. S., and Carvill, M.: Anterior Ocular Tuberculosis, Tr. Am. Ophth. Soc. **25**:104, 1927.

16. Fleisher, B.: Zur specifischen Behandlung von Augentuberkulose, Klin. Monatsbl. f. Augenh. **77**:47, 1926.

figures are as follows: Among the thirty patients with iridocyclitis who were treated with tuberculin seventeen were cured, three had recurrences, nine were improved, one was not improved and two were still under treatment. Among the eight patients with sclerosing keratitis and scleritis, four were cured, three improved and one had a recurrence. Among the patients with choroiditis, one was cured, three were improved and in one case the treatment was a failure. The cures, he believed, were due to the tuberculin and especially to Tebeprotin of Toenniessen.

During the past year, Woods and Rones [17] reviewed a series of forty-two cases of ocular tuberculosis in which tuberculin was used. The summary is as follows: In 45 per cent of the patients treated, the condition appeared to be healed, in 45 per cent there was definite improvement and in 10 per cent no improvement; 25 per cent of the patients treated showed recurrences of the local disease. The highest percentage of good results was observed in posterior ocular tuberculosis; in 61 per cent of the cases there was definite healing with no evidence of activity, and in 30 per cent there was definite improvement.

Ten years ago, Irvin Török [18] read a paper before the New York Academy of Medicine, giving the results in 100 patients with ocular tuberculosis treated with tuberculin. His figures were based on a clinical experience of fourteen years at the Knapp Memorial Eye Hospital. Among eight cases of interstitial keratitis, there were eight recoveries without recurrence. Thirteen of the twenty-one patients with scleritis were observed over a long period of time, with one failure; six had a relapse. Eight patients disappeared from observation prematurely; four of these recovered and four were improved. Only two of twelve patients with iridocyclitis recovered with no recurrences over a period of ten or eleven years; five showed no benefit and five improved. Török noted a therapeutic effect in five patients with heterochromic cyclitis. All showed a focal reaction. Three patients with chorioretinitis were under observation for four years; two recovered and one was improved. Three of the four patients with choroiditis disseminata recovered and one was not benefited. The observation period was from three to six years. The eleven patients with periphlebitis retinae were under treatment for from seven months to two years. All recovered with no relapse in from three to six years, except in one case. Török concluded that tuberculin is a valuable remedy in the treatment for ocular tuberculosis. He advocated long continued treatment. He considered positively tuberculous only those cases in which a positive focal reaction was obtained. The statistics

17. Woods, A. G., and Rones, B.: The Therapeutic Use of Tuberculin in Ocular Tuberculosis, South. M. J. **21**:613, 1928.

18. Török, E.: Tuberculin in the Diagnosis and Treatment of Eye Diseases, Arch. Ophth. **48**:242, 1919; discussion, p. 295.

referred to are given in the accompanying table. For various reasons, fifteen cases of Török are excluded, also, as mentioned, some of Fleisher's.

Rollet and Collrat,[19] authors of a recent French treatise on ocular tuberculosis, are extremely skeptical as to the value of tuberculin therapy in tuberculosis of the eye. They challenged the statistics of von Hippel, Bernheimer, Hertel, Herrenschwand and Weigelin on the grounds that the tuberculous nature of the ocular disease appearing in the statistics was not positively determined and because the part played by spontaneous recovery was not properly appreciated. These objections do not apply to the statistics of Hertel, who insisted on the necessity of taking spontaneous cures into account and who used all available means, both local and general, to establish the tuberculous nature of the conditions studied. Among his second group of patients in whom tuberculin acted favorably, some had been treated nonspecifically previously, with abso-

Statistics on Cases Reported in the Literature

Authors	Number of Cases	Healed, per Cent	Improved, per Cent	Unimproved, per Cent
Von Hippel	2'3	75.7	17.3	7.0
Weigelin	77	41.6	34.0	24.4
Hartig	38	26.3	42.0	31.7
Fleisher	43	51.0	35.0	4.5
Woods and Rones	42	45.0	45.0	10.0
Török	85	67.0	17.5	15.5

Hertel: 29 per cent more patients healed with specific than with nonspecific methods.

lutely no result. The comments of Rollet and Collrat appear in the chapter on tuberculosis of the uvea and apply particularly to that type of ocular involvement. In recording their personal experience, they objected to treatment with tuberculin because of the uncertainty of the dosage and because of the reactions produced; also because it may, in their opinion, sometimes act injuriously even when cautiously used. They stated, however, that they were not willing to go so far as to say that tuberculin has never effected a cure in tuberculous uveitis. They did not analyze the statistics of Török, which included only cases in which a focal reaction had been obtained, although they referred to them.

Attempts have been made to determine the effects of tuberculin in experimental tuberculosis of the iris. Schieck could find no difference in the cases of experimental tuberculosis of the iris in which tuberculin was used and in those in which it was not used. Krusius could not determine a definite, beneficial effect in tuberculin inoculation keratitis. Hertel [6] summarized the results of these investigations and concluded that it has not been possible to effect a cure with tuberculin in ocular

19. Rollet, E., and Collrat, A.: Tuberculose de l'oeil et de ses annexes, Paris, Gaston Doin, 1927.

tuberculosis experimentally produced by inoculation. It is impossible to derive any significant inference in regard to ocular tuberculosis in man from such experiments. Ocular tuberculosis in man is usually due to metastasis, and the histologic variation in the ocular lesion resulting is consequently dependent on the allergic state of the patient at the time of such metastasis. There is no parity between primary inoculation in a noninfected animal and ocular tuberculosis in man as ordinarily encountered. Also, ocular inoculation through the blood stream differs radically in its results from direct inoculation. The number and the virulence of the bacilli, the existing state of relative immunity, as well as the differences of the host, tend to vitiate the dependability of the conclusions which may be drawn as to the effect of tuberculin in experimental tuberculosis. It is not my wish to imply that no useful information is to be obtained from experiments on animals which may be applied to tuberculosis in man in general, but rather I wish to indicate the difficulty that makes for inaccuracies in experimentally produced ocular tuberculosis in regard to the effects of tuberculin.

My own experience is not of sufficient duration or extent to furnish statistics as to the ultimate effect of tuberculin in a large series of cases of ocular tuberculosis followed over a long period of years. Nor are reliable statistics as to these points available in the institutions with which I am connected. My material embraces thirty-seven cases from my private practice, some of them observed for years. These cases are exclusive of patients observed in public hospital practice or in consultation, as I did not have such patients under my personal supervision constantly. Also it does not include studies made on the effect of tuberculin in ocular tuberculosis in the Meller Clinic in Vienna. The only personal contribution that I can make to the subject is a statement based on convictions forced on me by a study of the more immediate effects and the value of tuberculin in this clinical material. A fundamental question to which I have attempted to find an answer so far as my personal practice is concerned is, Has tuberculin any practical value in ocular tuberculosis? To the enthusiast as to its favorable effects, this question appears superfluous; but as stated, eminent authorities in ophthalmology have denied its value.

As a criterion of the success of tuberculin in pulmonary tuberculosis, Pel [20] formulated the following conditions: No improvement in the patient's condition shall have occurred under adequate hygienic, dietetic treatment in the proper environment; then specific therapy is to be undertaken. If an improvement in the local condition is immediately apparent, the success of tuberculin therapy so far as the immediate condition is concerned can be regarded as proved. The same criterion may be

20. Pel, P. K.: Die Tuberculin Behandlung der Lungentuberculose, Berl. klin. Wchnschr. **46**:1717, 1909.

applied to the eye, with the additional demand that local and general therapy had been employed without avail before tuberculin was employed. A report of the first case that could come under this category is as follows:

CASE 1.—J. S. was first seen in 1917, when a diagnosis of scleritis of the left eye was made. The condition cleared up under the usual methods of therapy in several weeks. There was a recurrence of the scleritis in the same eye in 1923. Results of repeated examinations were negative, except for evidence of old arthritis in the right limb and of scars in the submaxillary region due to previous lymphatic involvement. Under the usual methods of treatment, the disease improved and then recurred with remissions and exacerbations. The patient was disabled for months and was unable to carry on her work as a dressmaker. In June, 1924, she was given tuberculin for diagnostic purposes. The skin and general reactions were positive. She was treated by the injection of tuberculin T. R., subcutaneously, beginning with 0.001 mg. Rapid improvement in the scleritis followed. No ocular reaction took place, but skin and general reactions followed each injection. The physical examination of the lungs revealed no evidence of tuberculosis. Doubtless, the primary complex of Ranke was present; as is often the case, however, it could not be demonstrated by physical examination. In September, 1924, the patient resumed her occupation of sewing. The injections of tuberculin were kept up for months after all symptoms had subsided. When the patient was seen within the past two weeks, the eye was entirely free from all signs of inflammation and had given no trouble since 1924.

CASE 2.—F. M., a man, was first seen in February, 1924, because of recent iridocyclitis in the right eye. There were sequelae of iridocyclitis in the left eye. The patient was referred to the eye service of the general hospital, where he received routine treatment. He was not cared for personally, as I was not on duty at the time. The acute process subsided. He was carefully examined generally, and it was thought that focal infection from bad teeth was the causative factor. Betterment in the ocular condition was ascribed to the extraction of the diseased teeth. He disappeared from observation and was not seen again until May, 1926. The condition in the eyes was quiet, but the changes in the iris were characteristic of tuberculosis. He was admitted to the Jewish Hospital for a von Pirquet test. This test was positive; following it, he had an ocular reaction. He left the hospital on his own initiative and was not seen until the next fall. He returned with the complaint of pain and irritation more pronounced in the right eye, which disabled him almost continually. The patient again was sent to the Jewish hospital on my service. At that time, complete physical examination revealed a hyperplastic rhinitis, a deviated septum and gallstones. There was no clinical evidence of pulmonary tuberculosis. An operation on the nose relieved the nasal condition but had no effect on the iridocyclitis. A von Pirquet test gave a positive reaction. Following this test, the left eye showed a reaction as on a former occasion. Injections of tuberculin bacillus emulsion of 0.00001 mg. were given and gradually increased. In January, 1927, the left eye showed a reaction following an injection. There was no skin or general reaction. This reaction had a favorable influence on the disease; the appearance of the iris, observed with the slit-lamp, showed rapid improvement. The improvement continued, and the patient was discharged in March, 1927, with nearly normal vision in each eye, the eyes being free from all irritation or pain. Shortly after this time, the patient returned to his home in Germany. He was examined, at my request, in January, 1929, by Dr. A. Pilzecker, an ophthalmologist in his home town, who reported that his eyes, at the time of writing, were quiescent and that the patient had been able to follow his occupation

since 1927. In May, 1927, his left eye became irritated, owing to outdoor exposure connected with his work. Since carrying on his work indoors, he has had no further trouble.

CASE 3.—N. H., a woman, aged 28, was first seen in 1920, when a diagnosis of chronic iridocyclitis in the left eye was made. The Wassermann reaction was negative. The condition was ascribed by me to focal infection due to poor teeth and ethmoiditis. Healed pulmonary tuberculosis involving the lymph nodes was found. Other physical observations were without significance. Under local treatment and removal of focal infections, the condition improved. Recurrences took place, for which she was treated by local methods. In June, 1925, a von Pirquet test was made which gave a strongly positive reaction. For several months she was given treatments with tuberculin, varying in dosage from 0.001 to 0.5 mg. of tuberculin T. R. The condition of the eye improved. The injections of tuberculin were discontinued in January, 1926. When the patient was seen in January, 1929, the eye was free from inflammation. She had normal vision. There were quiescent evidences of previous iridocyclitis.

CASE 4.—A. D., a woman, aged 31, was first seen in April, 1923, at which time she had a deep marginal keratitis of the right eye. Physical examination gave negative results except for evidences of intestinal intoxication. There was no evidence of focal infection. Under local treatment and attention to the intestinal tract, especially by placing the patient on a nontoxic diet, the condition subsided gradually. It was felt at the time that the case was significant as indicating the influence of intestinal intoxication on the eye. The patient returned in March, 1926, with episcleritis. Nodules were observed in the iris in the ciliary portion; also, fish-spawn or sugar candy, ill defined exudates were seen in grazing illumination of the slit-lamp. Treatment with tuberculin was begun with a dosage of 0.1 cc.; 0.001 of tuberculin T. R. and 0.1 cc. of tuberculin B. E. The skin reaction was always extensive and pronounced. There were no general or focal reactions. Three weeks after beginning treatment, a definite improvement was obvious. The eye was less injected. A nodular area in the iris had diminished. The exudates were less extensive. The injections of tuberculin were continued over a period of months. She was discharged with normal vision and resumed her occupation as bookkeeper; she had been free from disability up to January, 1929.

COMMENT

These patients were in a state of more or less continuous disability, and frequent interruptions in their work had resulted in great economic distress to them and their families. After the specific treatment, early improvement was observed. The treatment was kept up after the condition of the eyes had become quiescent.

I have selected these cases because they could be subjected to the criterion mentioned and also because in their selection I seem to avoid the fallacy to which attention was directed by Hertel,[6] Derby,[15] Rollet and Collrat,[19] among ophthalmologists, and Neumann [21] among internists,

21. Neumann, Wilhelm: Beiträge zur spezifischen Behandlung der Tuberkulose auf Grund klinischer Beobachtungen, Beitr. z. Klin. d. Tuberk. **39**:163, 1918; Ibid. **17**:69, 1910; Beiträge zur spezifischen Behandlung der Tuberkulose: III. Med. Klin. **21**:1844, 1925; Der Wert der Tuberkulinbehandlung; Ibid. **23**: 1129, 1927.

of assigning to treatment with tuberculin favorable effects in cases when tuberculin had been early employed but in which spontaneous cures might have occurred. Besides, it is not necessary to multiply the number of cases indefinitely to sustain the modest assertion that tuberculin cannot be rejected as valueless.

I cannot refrain at this point from giving the report of a case taken from a paper by Professor Neumann [22] of Vienna, in 1927, on the value of tuberculin therapy. This eminent authority on the use of tuberculin and on tuberculosis in general classified various types of pulmonary and nonpulmonary tuberculosis in which he believed tuberculin therapy to be indicated. The seventh of his classifications concerns ocular tuberculosis, and it is particularly significant as he is an internist. The translated report of the case is as follows: Neumann saw the patient in June, 1926. The history revealed that he had consulted an ophthalmologist in Vienna two years previously because of scleritis and choroiditis. The internist called in could not discover any tuberculous pulmonary lesion, and ascribed the ocular disease to uric acid diathesis. The patient was given, therefore, a purine free diet and local treatment. The condition progressed despite treatment and terminated in the total loss of vision in the eye, which remained painful. For this reason the patient returned to Vienna. He was examined by Professor Neumann, who found a pronounced postpleuritic tuberculosis fibrosa sinestra. Treatment with Toenniessen's Tebeprotin was instituted, and three months later the patient was free from all pain in the blind eye and the inflammation had subsided, while the vision in the fellow eye had decidedly improved. Appearing as it did in a general paper, this report furnished no detailed ophthalmologic data, but the marked benefit in the ocular condition under tuberculin therapy when all other efforts were in vain, is obvious. Neumann's concluding remarks are interesting and refer definitely to ocular tuberculosis, among other conditions. A translation is as follows: "I have presented only certain definite types of tuberculosis in which, in the light of my twenty years' experience, tuberculin is pre-eminently effective, so pre-eminently that upon the basis of this long experience, I should like to directly characterize a failure to give it a trial in the above mentioned types of tuberculosis as culpable negligence." In fact, he used the word "Kuntsfehler," which means malpractice.

Will the use of tuberculin diminish or prevent the number of recurrences in ocular tuberculosis? My experience convinces me that in a certain proportion of cases it will do so. I am certain that after treatment with tuberculin a large proportion of my patients have been able to pursue occupations involving strenuous use of their eyes, whereas

22. Neumann (footnote 21 fourth reference).

before, they were disabled by frequent recurrences. Hertel [6] seemed properly to value tuberculin in a survey previously given when he subdivided the cases into three groups: (1) those in which tuberculin is of no value; (2) those in which it acts favorably, and (3) those cases in which spontaneous cure takes place and in which tuberculin is devoid of influence.

An imposing array of authorities have ascribed great value to tuberculin. It is noteworthy that the authors of three recent monographs in German on ocular tuberculosis, Löwenstein,[7] Bergmeister [23] and Werdenberg,[24] were unanimous in endorsing its use in ophthalmology.

Among American authors, not already mentioned, who recently reported concerning the use of tuberculin in ophthalmology are Weeks,[25] Wilmer,[26] Hill,[27] Peter,[28] Luedde,[29] Finnoff [30] and Kiefer and Shulman.[31] Dr. John E. Weeks advocated tuberculin therapy in a paper appearing in 1918 and in another publication in 1926, and he insisted on its use even after all symptoms have disappeared. Fleisher, in a recent article, referred to this attitude of Weeks and highly commended it, as he stated the belief that the use of tuberculin over a long period of time is necessary to prevent recurrences. Peter [28] made the following statement in his paper: "The citation of case histories will not add to the purpose of my paper. Tuberculin needs no defense. It is a product of admittedly great merit and in harmony with the trend of modern medicine."

An inquiry into the value of tuberculin may justifiably be concerned with the efforts that are now being made to define more definitely its indications and contraindications with the hope of enhancing its usefulness in the future. Werdenberg [24] has recently attempted to formulate indications and contraindications for the use of tuberculin based on the

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23. Bergmeister, Rudolf: Tuberkulose Erkrankugen des Auges, Berlin, S. Karger, 1927.

24. Werdenberg, E.: Theorie und Praxis der Augentuberkulose, Stuttgart, Ferdinard Enke, 1926.

25. Weeks, J. E.: Tuberculosis of Eye, Am. J. Ophth. **9**:243, 1926.

26. Wilmer, W. H.: Clinical Aspects of Ocular Tuberculosis, Arch. Ophth. **57**:1 (Jan.) 1928.

27. Hill, Emory: Tuberculosis in Relation to the Eye, South. M. J. **21**:607, 1928.

28. Peter, L. C.: The Use of Tuberculin in Ocular Tuberculosis, Tr. Acad. Ophth. & Otolaryng., 1917-1918, p. 451.

29. Luedde, W. H.: The Significance of the Tuberculin Reaction and Other Problems in Ocular Tuberculosis, Am. J. Ophth. **6**:161 (March) 1923.

30. Finnoff, W. C.: Differential Diagnosis in Tuberculosis of the Eye, J. A. M. A. **90**:341 (Feb. 4) 1928.

31. Kiefer, H. A., and Shulman, Leon: Personal Observations on Tuberculosis of the Eye, Tr. A. M. A., Sec. Ophth., 1927, p. 128.

work of Ranke and its applications to ocular tuberculosis by Schieck and Gilbert and also on the modern conception of immunity in tuberculosis. Ranke [1] made the noteworthy assertion that it is no longer possible to speak of tuberculin therapy without a knowledge of the various stages of tuberculosis, and this statement most assuredly applies to the use of tuberculin in ocular as well as in general tuberculosis. Equally important is Ranke's dictum that the most important step in the understanding of tuberculosis is taken when one directs one's attention to the general picture of the disease of the total organism, not to the involved organ exclusively.

To the researches of Ranke one owes the conception that the histologic variation in tuberculous tissue is due to the native response of the tissues to the tubercle bacillus plus the effect of allergy. He formulated a useful, hypothetic classification of tuberculosis into a primary stage with definite localization, accompanied at first by normal sensitization; a secondary state of generalization with hematogenous and lymphogenous metastases with an acquired hypersensitiveness, and a tertiary stage of isolated tuberculosis in one or more organs with a lessened hypersensitiveness, usually under the conditions unfavorable for metastasis. He did not imply, however, that the bodily organism necessarily must pass through all those stages.

With the exception of the rare cases of primary tuberculosis of the external parts or of the deeper ocular structures due to penetrating injuries, ocular tuberculosis always occurs as a result of a metastasis. As a matter of fact, the types of ocular tuberculosis usually encountered clinically in adults belong to the late second or third stage of relative immunity. They are predominately nodular. As a rule, in tuberculosis of the eye, only slight evidence of pulmonary tuberculosis is found by physical examination. Roentgen examination reveals usually only slight changes. Werdenberg's [32] examination of the lungs by the roentgen rays in ninety cases of ocular tuberculosis in which the pulmonary involvement was not pronounced revealed the following lesions: a clearly marked, usually double-sided primary complex; slightly increased irregular shadows of the hilum, and slight apex clouding on one or both sides as fine, disseminated shadows. Invariably there was evidence of mild and frequently old foci of hematogenous dissemination, usually localized in the lower lobe. Especially characteristic were the changes in the pleura. The x-ray picture was positive in all cases. The patients were mostly young adults up to the age of 40 years. The x-ray pulmonary observations of Neumann in cases of ocular tuberculosis are substantially in accord with those of Werdenberg. Severe ocular lesions

32. Werdenberg, E.: Ueber Lungenbefund bei Augentuberkulose, Klin. Monatsbl. f. Augenh. **78**:238, 1927.

were frequently accompanied by slight pulmonary involvement, and vice versa. The constitutional symptoms of tuberculosis bore, on the contrary, a direct relation to the character of the ocular disease. A slight pulmonary tuberculous involvement is regarded by Werdenberg as actually predisposing to ocular tuberculosis.

It may well be that slight, nonocular metastases may occur in pulmonary tuberculosis with negative or slightly positive clinical and x-ray observations which entirely escape observation. A minute lesion in the eyes is likely to become obvious immediately because of its disturbance of function and its accessibility to observation. The x-ray observations mentioned would probably excite no comment if no ocular lesion existed, as they could be regarded as devoid of clinical significance. The increased appreciation among ophthalmologists of the importance of the primary focus will probably lead to the further perfection of x-ray technic in the reliable interpretation of the finer details of the pulmonary lesion.

Werdenberg [24] regarded tuberculin as contraindicated in ophthalmology in the highly allergic, exudative lesions in the stage of hypersensitiveness, because of the danger of injurious local and general reactions. He believed it to be indicated in the frequent nodular, proliferative, slightly allergic lesions with diminished hypersensitiveness. It has been pointed out, however, that in tuberculous lesions in general an exudative, allergic process may be beneficent in its tendencies; a nodular, proliferative process may terminate disastrously from the standpoint of the destruction of tissue. From a purely ocular point of view, exudation with its consequences is more likely to be destructive of function than proliferation. Neumann [22] held tuberculin to be especially indicated in some forms of serosanguineous pleuropneumonia which is predominately exudative, and therefore a highly allergic process. Löwenstein [7] stated that he had not been impressed with a radical difference in the healing tendency in exudative and nonexudative processes. Neumann [22] made the statement, which is startling to an ophthalmologist, that the clinician who uses tuberculin only when it is well tolerated knows but little concerning its proper employment. Werdenberg [33] presented a paper on the subject of the relation between ocular and general tuberculosis at the last meeting of the Deutsche Ophthalmologische Gesellschaft in Heidelberg. He pointed out that for the ophthalmologist the qualitative aspect of the local and general disease, that is, the toxicity and the intensity of the process, is more important as a basis for specific therapy than the quantitative, the extensive features of the pulmonary

33. Werdenberg, E.: Ueber Beziehungen der Augentuberkulose zur Tuberkulosen Algemeinerkrankung, abstr. in Klin. Monatsbl. f. Augenh. **81**:328, 1928.

or ocular lesion. He promised a publication of an article dealing with a modification of the valuable classification of pulmonary tuberculosis by Turban [2] to meet the needs of the ophthalmologist in specific therapy. Stock, in discussing this paper, stated that he did not believe that the indications and contraindications could be so sharply defined that in one case one could say it is blameworthy to use tuberculin and in another to apply roentgen therapy. He maintained that in the therapy of tuberculosis one must resort to empiricism and that the competent clinician intuitively will institute the proper treatment after careful observation. One must feel that there is much truth in this observation of Stock, but at the same time one cannot help but commend heartily the efforts of Werdenberg to minimize the purely empiric in specific therapy.

The cause of all the controversy, misunderstanding and misinterpretation as to the value of tuberculin arises from the nature of the disease, tuberculosis, a disease which is protracted, protean in its manifestations, governed by a resistance which is relative, variable and subject to incalculable influences and which presents many features which are paradoxic, a disease the outcome of which cannot readily be predicted, and the pathologic background of which seems at first glance to be chaotic. This confusion will be dispelled as the knowledge of tuberculosis progresses. In the meantime, one must rely mainly, for practical purposes, on clinical observation.

An account of two cases selected from the literature will illustrate the truth of this statement. The first case is one reported by Hertel.[6] It was a case of severe iridocyclitis in which no specific treatment was carried out and which terminated in seclusion of the pupil with complete blindness in one eye. The fellow eye displayed fresh precipitates, nodules in the angle of the chamber, exudates in the pupil and opacities of the vitreous. Following the institution of tuberculin therapy, the iridocyclitis in this eye subsided, and useful vision was obtained. The second case reported by Rollet and Collrat [19] was one of conglomerate tubercle of the iris with caseation. Following an injection of 0.00001 mg. of tuberculin, a general reaction occurred and the lesion in the iris became confluent. The fellow eye, unaffected up to this time then showed pericorneal injection accompanied by photophobia and ciliary pain. The eye originally affected had to be enucleated. The other eye developed a tuberculous keratitis. Spontaneous recovery occurred in this eye at the end of six months.

These cases are interesting not so much because they reveal the possibilities of error in attributing to the action of tuberculin, a cure which could be due to spontaneous recovery, but because they emphasize the importance of the rôle of the primary focus and the state of relative immunity in any healing process which may ensue. Doubtless in both

these cases the subsidence of the inflammation in the second eye was due to the development of a beneficent state of relative immunity accompanied by quiescence or healing in the primary focus. There is no doubt that recovery, either spontaneous or induced by tuberculin, may occur in ocular tuberculosis. There is a point made by Schieck, in discussing Fleisher's [16] paper on Toenniessen's tuberculin, which is significant in connection with the question of recurrences. Schieck stated that, in his opinion, recurrences in healed ocular tuberculosis implied new metastases. Fleisher concurred in this opinion.

In considering the prospects of enhancing the value of tuberculin therapy in the future, attention must be given to a new tuberculin, that of Toenniessen.[34] I am aware that a host of new tuberculin preparations have been lauded by their originators as the ultimate panacea. Unfortunately, these claims have often been discredited by subsequent experience. There is distinct evidence, however, that Toenniessen [34] has produced a tuberculin which has distinct advantage over those hitherto employed. He has attempted to simplify the complex mixture which exists in the tuberculins previously used, a mixture in which the proportions of the components are uncertain and not susceptible of exact control. The result of his efforts has been Toenniessen's Tebeprotin which, he claims, as the result of experiments on guinea-pigs, is ten times less toxic than old tuberculin with a much greater specific effectiveness. These qualifications increase its value for diagnostic purposes as the equivalent of a dangerous dose of old tuberculin can be given with a minimum risk and with enhanced specific action. It is said to be a pure, chemical substance, soluble, uniform and susceptible of exact dosage. The diagnostic dose is 0.1 mg. The initial therapeutic dose is 0.001 mg., or less, according to indications. One great advantage of the preparation is that the usual maximum dosage of 0.1 mg. can be maintained in subsequent treatment and does not have to be increased. The preparation is extremely convenient to use, as two bottles containing dilutions numbered 1 and 2, which preserve their effectiveness for a year, are all that is ordinarily required. The preparation has been highly endorsed by Fleisher,[16] Urbanek [35] and Asmus [36] among ophthalmologists. It is in routine use in the Meller Clinic. I have used it in ten cases, and

34. Toenniessen, E.: Die spezifische Erkennung und Behandlung der Tuberkulose mit einem aus Tuberkeibazillen gewonnenen Eiweisskorper, Deutsche med. Wchnschr. **50**:629, 665 and 708, 1924; Ueber die Verwendung eines aus Tuberkelbazillen gewonnenen Eiweisskorpers zur spezifischen Diagnostik der Tuberkulose, ibid. **69**:957, 1922.

35. Urbanek: Erfahrungen mit dem Tebeprotin Toenniessen, Verhandl. d. deutsch. ophth. gesellsch., Heidelberg, 1925.

36. Asmus, Edward: Zur Behandlung der Augentuberkulose mittels Tebeprotin Toenniessen, Klin. Monatsbl. f. Augenh. **80**:302, 1928.

while it is impossible to draw final conclusions from such a small material, I am inclined to believe that the claims of Toenniessen and of the ophthalmologists mentioned are well founded. The elimination of the complex factors present in other tuberculins may aid greatly in simplifying the evaluation of tuberculin as a therapeutic agent in future statistical investigations.

It is not possible within the limits of this paper to enter into the question of the diagnostic value of tuberculin. This subject has been fully dealt with by Dr. W. H. Luedde, of St. Louis, in a paper entitled, "The Significance of the Tuberculin Reaction and Other Problems in Ocular Tuberculosis," which appeared in 1923.

Ophthalmologists in this country are deeply indebted to Dr. Derby and Dr. Carvill [15] for their study of a series of cases of anterior segment tuberculosis, which leads them to the conclusion that tuberculin is, in the long run, practically valueless. Too great a complacency has prevailed perhaps as to the results of treatment with tuberculin. Their critical attitude will do much good and prevent much harm. Certainly, the statistics of the past could be improved on. Doubtless, their work will lead to a revision of the entire subject and to the initiation of a movement for the uniform compilation of statistics in which the sources of error will be recognized and eliminated as far as possible. A noteworthy effort has been made by Woods and Rones [17] in this direction in their statistical study of the effects of tuberculin in the material of the Wilmer Institute.

CONCLUSIONS

1. The preponderance of evidence indicates that tuberculin is of great value in the treatment for ocular tuberculosis.

2. In ocular tuberculosis, the attitude of the ophthalmologist that he is dealing, except in primary ocular tuberculosis, with a tuberculous subject who has a secondary incidental ocular localization will be fruitful in the future in enhancing the value of specific therapy in the cure of the ocular lesion as well as in the prevention of recurrences, which depends on rendering the primary focus inactive under the influence of a favorable state of relative immunity.

3. The further compilation of more reliable statistics is desirable.

4. Personally, I feel compelled to take my stand with Ranke, who believes that further progress in tuberculin therapy is only a matter of time, and with Neumann, who contends that failure to use it in properly selected cases will result in avoidable loss of sight and needless suffering.

Notes and News

EDITED BY DR. GEORGE S. DERBY

SOCIETY NEWS

Program for the Academy of Ophthalmology and Otolaryngology.—The scientific program of the next meeting of the Academy of Ophthalmology and Otolaryngology, which is to be held at Atlantic City next October, will be as follows:

JOINT SESSION

Address by the president.....Dr. Harris P. Mosher, Boston
Address by the guest of honor.....Mr. Herbert Tilley, London, England
Symposium on Brain Abscess:

1. From the Standpoint of the Ophthalmologist.....Dr. W. L. Benedict, Rochester, Minn.
2. From the Standpoint of the Otolaryngologist.....Dr. Wells P. Eagleton, Newark, N. J.
3. From the Standpoint of the Brain Surgeon..Dr. William J. Mixter, Boston
4. Methods of Drainage.....Dr. Harry P. Cahill, Boston

Discussors: Dr. George W. Swift, Seattle, and Dr. S. J. Kopetsky, New York

OPHTHALMOLOGIC PROGRAM

1. Contracted Visual Fields: An Analysis of Two Hundred and Fifty Consecutive Cases..Dr. W. D. Rowland and Dr. A. W. Rowe, Boston
2. Pathology of Perforating Injuries of the Eye with Report of Case .....Dr. Georgiana Dvorak Theobald, Chicago
3. Conservation of Lacrimal Sac.....Dr. F. N. Knapp, Duluth, Minn.
4. The O'Connor Shortening Operation..Dr. Joseph L. McCool, Portland, Ore.
5. Removal of Number Eight Lead Shot from the Vitreous with Resultant Vision of 6/6. Case Report.....Dr. George H. Cross, Chester, Pa.
6. Acute Erythema with Stomatitis and Ophthalmia.....Dr. John M. Wheeler, New York
7. Eales Disease.....Dr. Charles A. Young, Roanoke, Va.
8. Report of Research Fellow in Ophthalmology.....Dr. D. B. Kirby, New York
9. Accessory of Effects of Correcting Glass by Its Position Before the Emmetropic Eye.....Dr. Alfred Cowan, Philadelphia
10. Tuberculosis of Iris Following Extraction of Cataract. Case Report .....Dr. Nelson M. Black, Milwaukee

Cleveland Ophthalmological Society.—At the first dinner meeting of the Cleveland Ophthalmological Society, Dr. Clarence King of Cincinnati spoke on "Some Instruments and Procedures of Value in Cataract Extraction."

The organization of the society was completed by the election of the following officers for the ensuing year: president, Dr. W. E. Bruner; vice president, Dr. H. H. Shiras; secretary-treasurer, Dr. M. Paul Motto.

Ohio State Medical Association.—At the meeting of the Ohio State Medical Association, Dr. John M. Wheeler, professor of ophthalmology at Columbia University, was the guest of honor. He spoke to the ophthalmic section on "Exophthalmos from Different Causes."

Los Angeles County Medical Association.—A symposium on bronchial asthma was presented by the eye and ear section of the Los Angeles County Medical Association on April 1, by Drs. Burton D. Haseltine, Alvin W. LaForge and Alonzo H. Waterman, all of Chicago, and Dr. Dean W. Myers of Ann Arbor, Mich.

Chicago Ophthalmological Society.—The Chicago Ophthalmological Society was addressed on May 27 by Drs. George W. Hall and Roland P. Mackay at the Medical and Dental Arts Club, on ophthalmoplegic migraine, and by Dr. George F. Suker, on idiopathic hypertension and the ophthalmic observations.

"EYE SPECIALIST" SENTENCED

The California State Board of Medical Examiners reports that one William McBride pleaded guilty on March 7 to practicing without a license, and was sentenced by Judge Finney at El Centro to serve six months in jail. McBride was arrested for performing a fake "radium operation" to remove a cataract from the eye of James Cook, an aged man living near Westmoreland in Imperial County. He was accompanied by Roy E. Francis who had a membership card of the International Alliance of Theatrical Stage Employees and Moving Picture Machine Operators of the United States and Canada. The part Francis played was to tell the victim that McBride was a great eye specialist. McBride, at the time the offense was committed, was on parole from a penitentiary sentence in Oregon. He practiced similar fake operations there and was eventually indicted and convicted for larceny. His method in Oregon was to locate some aged person who had trouble with his eyes and who had money. An assistant would call on the prospect, advising him to go to a distant city for radium treatment to save his sight. The victim objected, naturally, and he was then told of the great eye specialist, McBride. McBride would put something in the victim's eye which would coagulate, then pull it out with tweezers, show it to the victim, collect a fee at once and leave. It is reported that McBride's wife made restitution to all persons in Tallamook County, Oregon, from whom her husband thus obtained funds, and paid the costs of his prosecution. It was under those circumstances that his Oregon sentence was postponed and McBride was permitted to go to California.

GENERAL

Dr. W. E. Bruner, professor of ophthalmology in the Western Reserve University School of Medicine, has left for a three weeks' holiday at his camp in the Adirondacks.

Dr. Ernest H. Rowen was recently appointed resident ophthalmologist in the ophthalmic division of Lakeside Hospital.

DEATHS

Giuseppe Cirincione of Rome, one of the best known of Italian ophthalmologists, and editor of the *Annali di ottalmologia,* died on March 19, 1929.

Filippo Speciale, professor at Turin, and collaborator with Professor Cirincione on the *Annali di ottalmologia,* died in January, 1929.

JAMES MOORES BALL

1862-1929

UNIV. OF MICH.

Obituary

JAMES MOORES BALL, M.D.
1862-1929

Dr. James Moores Ball, prominent ophthalmologist of St. Louis, died of heart disease on March 1, 1929, at his home in that city.

He was born at West Union, Iowa, in 1862. He received his degree in medicine from the University of Iowa, and postgraduate studies were pursued in this country and abroad. The chair of ophthalmology in the old College of Physicians and Surgeons in St. Louis was occupied by him, and he held appointments on the staffs of various hospitals during his lifetime. Many prominent oculists and physicians of today attended his lectures. In recent years he again came into prominence as a lecturer on medicohistorical subjects, appearing before postgraduate classes in many cities; in this field he attained a unique position, owing to his untiring and productive efforts in research. At various times he served on the editorial staffs of scientific and medical publications.

As an author, he was internationally known for his contributions to ophthalmology and medical history; his "Modern Ophthalmology" is a standard and authoritative work on diseases of the eye. Another work, "Andreas Vesalius, Reformer of Anatomy," published in 1910 for private distribution, is an intimate picture of the man who, born in Brussels in 1510, first placed the science of anatomy on a definite basis. Last year saw the publication of "Sack-em-up Men," a gruesome account of grave robbing and murder to obtain material for anatomic study by surgeons in the universities of England, Scotland and Ireland. Several unfinished works by Dr. Ball remain, among which is a treatise on "Art and Anatomists" which lacks only two chapters of being complete. Up to a few days before his death, the hand of Dr. Ball continued to correct and edit the proofs of this, his last work.

While his professional obligations occupied most of his time, his chief diversions were his library and his friends, and he displayed always a reverence for both. During his career, he gathered much ophthalmologic material; this collection was given to the Army Medical Museum in Washington, where it now is housed in the James Moores Ball Room. The room is filled to overflowing with pathologic specimens, plates, engravings, charts and paintings of diseases of the eye, along with books, letters and autographs from many celebrated physicians and surgeons from all over the world. A portrait in bronze of the donor occupies a place with the collection.

Recently, Dr. Ball's rare and valuable collection of several hundred old medical volumes, which he gathered over a course of years, were donated by him to the St. Louis Medical Society. These books are now permanently housed in the society's library and they include first editions dating back to 1400.

His was a character dignified and unique in its friendships, and loyal in its affection. Those who knew him intimately can but look back and recall the unassuming worth of the man, the modesty of his speech and his kindly deference to the ideas of others. Always a teacher, to those who were willing to absorb from his years of experience he gave freely of whatever was contained in the vast storehouse of knowledge he possessed. New fads did not appeal to his sense of proportion, but he was always alert to seek and promote the better ideas which had definite merit.

His widow and their son and daughter survive him.

F. O. SCHWARTZ.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Anatomy and Embryology

CLINICAL AND ANATOMIC CONTRIBUTION TO THE THEORY OF PUPILLARY FIBERS IN THE OPTIC NERVE. E. BODENHEIMER and H. KORBSCH, Arch. f. Ophth. **121**:46 (Oct.) 1928.

A case of tumor of the brain with bilateral secondary optic atrophy following choked disk showed light reaction but no light perception in the right eye, while with the left the patient was able to distinguish fingers in the upper temporal quadrant and also showed light reaction. Anatomic examination a few days later revealed a group of normal axis-cylinders in the dorsal and median parts of each optic nerve. The possibility could therefore be considered that these intact fibers were "pupillary fibers," thus accounting for the light reaction in the blind eye. The literature concerning the assumption of special pupillary fibers in the optic nerves was critically reviewed but no evidence of their existence was found. Therefore, the group of fibers that was found intact in this case was considered the rest of the only type of centripetal fibers that exist in the optic nerve; namely, fibers that carry the impulses to the pupillary center as well as to the psycho-optical center. To explain the clinical fact that in this case the pupils reacted while the visual cortex did not, it is assumed that the threshold of the two centers is different for the same kind of stimulus. Therefore, under the conditions of severe atrophy of both optic nerves it is possible that a stimulus is just strong enough to cause a reaction of the pupillary center, but is too weak to make the visual cortex respond.

P. C. KRONFELD.

Physiology

ON THE LATENCY OF NEGATIVE AFTER-IMAGES FOLLOWING STIMULATION OF DIFFERENT AREAS OF THE RETINA. R. S. CREED and RAGNAR GRANIT, J. Physiol. **66**:281 (Nov.) 1928.

Experiments are reported which deal with the latency of after-images of objects situated in different parts of the visual field. White circular disks provided with a fixation point were observed binocularly under standard conditions of illumination, background and state of adaptation of the eyes. After a definite period of fixation a screen of the same color as the test object was lowered in front of the object, and the observer rapidly directed his gaze on a fixation mark in the middle of the screen so arranged as to be directly over the mark previously fixated on the disk. The time of the first appearance of the after-image was noted as it appeared projected on the screen. The authors found that the latency of the after-image was longest in the fovea centralis, and that on passing out toward the periphery it drops in an almost perfect curve to a point about 1 degree and 50 minutes of the visual angle. There then occurs a rather sudden lengthening of the latent period, reaching a maximum at about 2 degrees and 30 minutes from the fixation point. Passing further out toward the periphery, there is a second shortening of the latent period which occurs in the form of a hump. The position of this hump varied in different persons and in the same person slightly from

day to day but was always between 2 and 3 degrees from the fixation point. Curves representing the change in the latent period of the after-images from the fixation point out to the periphery were the same in all meridians of the retina.

The authors point out that the transitional area in the curves appears at a visual angle where the retina changes its structure from that of an organ dominated by cones to a rod organ. Outside an area subtending about 4 degrees of the visual angle (corresponding to 2 degrees of the abscissa in the curves presented in the paper), the rods suddenly come into play in greater force than in the cones. The sudden rise in the curves probably indicates that a new organ is now reacting to the stimulus and that either the latency of the after-image is longer for an object falling on the rods than that of one falling on cones, or that some more complicated interaction takes place between the two structures which in itself leads to a lengthening of the latent period. Later on in the paper, the authors adduce evidence that such interaction does occur.

They believe that the initial steep fall in the curves is exclusively a cone curve and is indicative of functional differences between the cones in the center of the fovea and those further out. Using disks of varying size, they find that the latency of the after-image of the disk as a whole is that corresponding to the region of the retina on which the image of its edge falls. The latency shortens as the size of the disk increases when the center of the disk is the fixation point, but when the center of the image is outside the macula, the areal extent of the primary image is devoid of influence. The changes in latency with disks in the center of the field of vision is due, therefore, not to any effect of spatial summation but to the fact that the edges are falling successively on receptors of changing character.

F. H. ADLER.

METHODS OF RECORDING EYE MOVEMENTS. M. D. VERNON, Brit. J. Ophth. **12**:113 (March) 1928.

Vernon presents a review of the various methods that have been used to record movements of the eye since the work of Javal in 1878. The different methods are divided into three groups:

(*a*) Indirect methods depending on photography of the eye, of reflection from the apex of the cornea, or of some small object attached to the cornea.

(*b*) Indirect methods depending on the attachment of a capsule or recording lever to the cornea or eyelid, the movements of which may be recorded by direct tracing on a drum, or else photographically.

(*c*) Methods of direct observation.

Vernon concludes that the photographic method, as used by Dodge, C. T. Gray, and Miles and Shen, has proved the most satisfactory, though it is probably difficult to manipulate, and requires expensive apparatus. Direct observation through a telescope is useful for less detailed work.

A. COWAN.

EXPERIMENTAL VARIATIONS OF THE OCULAR TENSION IN RELATION TO THE PHYSICOCHEMICAL PROPERTIES OF THE VITREOUS BODY. E. REDSLOB and P. PEISS, Ann. d'ocul. **165**:1 (Dec.) 1928.

The authors recently showed that the swelling of the vitreous depended on its p_H and that the swelling diminishes most rapidly on the acid side. They concluded: (1) in the normal eye the maximum swelling is never attained and (2) that the swelling of the vitreous is extremely sensitive to any change in its p_H. The foregoing conclusions were determined on the enucleated eyes of cows. To

modify the tension on living eyes, different solutions were injected into the vitreous of rabbits. The tonicity was checked by tonometric readings.

The technic of injecting sterile solutions into the vitreous was as follows: A fine needle was inserted transversely through the sclera of a rabbit, care being taken to avoid the lens; no more than 0.1 cc. of a sterile solution was injected. The intra-ocular tension was taken immediately before the injection, ten minutes after, then every quarter of an hour for the first few hours and at longer intervals for the following two or three days.

The first solution injected into several rabbits was normal serum. The tension rose to 110 or 120, quickly fell again to 30 and then gradually became normal. Then solutions of hydrochloric and glycerophosphoric acids were tried. With these the intra-ocular tension rapidly dropped below the normal line and oscillated around 15 mm. of mercury for about eight hours, when a second drop occurred; this time it oscillated around 10 mm. of mercury for ten days. Mounting progressively, it attained the normal level on the thirteenth day.

Injection of sodium hydroxide caused a rise of tension lasting for several hours, when a second rise occurred. After this second hypertensive phase, there was usually one of hypotension. One rabbit developed acute glaucoma, and the tension remained high for four months.

Injections of different forms of gelatin, to modify the swelling of the vitreous by changing the iso-electric point, were next attempted. A rapid fall of the ocular tension below 10 mm. of mercury after twenty-four hours was observed. It remained between 5 and 10 mm. for from two to eight days, and gradually reached normal about four weeks after the injection.

H. W. SCARLETT.

Congenital Anomalies

PERSISTENT PUPILLARY MEMBRANE AND CONGENITAL ECTOPIA LENTIS. A. R. CREBBIN, Am. J. Ophth. **12**:87 (Feb.) 1929.

A boy, aged 11, is said to have been born with a "misshapened pupil." One brother has 4.50 degrees of hyperopia, and a cousin is blind from congenital retinal pigmentary degeneration. The right eye is slightly exotropic, and the pupil eccentric up and in. The left pupil is eccentric upward but less so than the right. Both irides are tremulous, and both anterior chambers irregularly deep. Across both corneas, above, is an opaque advance of the limbus. The right lens is luxated in and slightly down; the left, up and in. Each eye shows a cobweb-like membrane arising from the lesser circle of the iris, and containing an irregular hole corresponding to the undilated pupil. This membrane is more marked in the right eye. Some suspensory ligament fibers can be seen in the aphakic portion of the pupil.

W. S. REESE.

Refraction and Accommodation

DOES "SCHOOL MYOPIA" EXIST? H. BIRBAUM, Arch. d'opht. **46**:38 (Jan.) 1929.

The author believes that the evolution of refraction from the hyperopia present at birth corresponds to the elongation of the eye with growth. Much work has been done to explain how school work produces myopia without showing that this is the only cause. It cannot be proved that these children would not have been myopic without school work.

If it is true that myopia increases while at school it is also true that hyperopia diminishes. Consequently those eyes that are less hyperopic at birth have a greater chance of becoming myopic. On this ground an increase of from 1 to 2 diopters of myopia is no more a pathologic condition than a decrease of hyperopia from 4 to 2 diopters. The fact that myopia increases more than hyperopia decreases is a purely mathematical problem. A long eye which lengthens 40 per cent will be much longer proportionately than a short eye which elongates 40 per cent. The author reviews the evidence in favor of the ontogenetic evolution of myopia and refraction in general. In closing, he quotes Landolt, who suggested that the tendency to myopia can be looked on as a phenomenon of adaptation in the struggle for existence.

S. B. MARLOW.

THE SPOT OF MARIOTTE IN CASES OF AXIAL MYOPIA. M. HEDINGER, Arch. f. Ophth. **121**:126 (Oct.) 1929.

The blind spot of the myopic eye lies slightly closer to the fixation point when tested with the corrected glass than when tested without. The amount of this displacement depends on the strength of the concave glass and is due to the prismatic action of the glass. In almost two thirds of the cases the blind spot appears slightly larger if tested with the correcting glass, because the glass abolishes the circles of diffusion. The blind spot is always found enlarged if there is a circumpapillary atrophy of the choroid, but the amount of enlargement does not correspond with the size of the apparently atrophic area; it is rather determined by the degree or stage of atrophy. The cases described have large atrophic areas and only slightly enlarged blind spots, indicating that the retina is only slightly damaged in this region. The same cases may show a larger blind spot after a few months without much change in the ophthalmoscopic picture, indicating a complete atrophy of choroid and retina in that region.

P. C. KRONFELD.

Experimental Pathology

COPPER CATARACT. LODDONI, Ann. di ottal. e clin. ocul. **57**:28 (Jan.) 1929.

The author inserted small pieces of copper wire in the vitreous of four rabbits and one dog and removed the eyes after varying periods. After two months, a deposit of copper granules was found on the anterior and posterior lens capsule and in the cortex just under the capsule. After five months, the capsule was diffusely infiltrated with fine copper granules, the color fading out into the superficial layers of the cortex. The capsular epithelium was degenerated. After one year a marked greenish opacity of the lens had developed, denser in the central region with spokes of opacity radiating to the equator, as in the clinical picture of copper cataract, but the spokes were not so numerous. The same picture was produced by prolonged immersion of human lenses in 1:10,000 to 1:1,000 copper sulphate solution. The nucleus never showed copper, even after immersion for six months. After from two to six months, star-shaped opacities with rays corresponding to the suture lines were produced. The nucleus remained clear, was sharply separated from the greenish cortex, and gave negative tests for copper. Decapsulated lenses became greenish after ten days, but no copper reached the nucleus. Repeated injections of copper sulphate into the vitreous never caused the absorption of copper by the lens.

The author believes that the stellate appearance of the opacity is due to shrinking of the anterior capsule. In a case reported by the author, the copper

was deposited in a thin layer, 0.5 micron thick in the center, so that it was not opaque on ophthalmoscopic examination, but only when light was thrown on it from the side. Loddoni believes that the greater compactness of lens fibers in the nucleus prevents even the finest particles of copper from penetrating it.

S. GIFFORD.

EXPERIMENTAL AND HISTOLOGIC INVESTIGATION OF THE INFLUENCE OF HIGHLY CONCENTRATED ULTRAVIOLET LIGHT ON THE EYE OF THE RABBIT. M. BUECKLERS, Arch. f. Ophth. **121**:73 (Oct.) 1928.

Isolated ultraviolet rays in high concentrations (from 400 to 285 millmicrons) cause only transitory and superficial lesions of the eye. Combustions of the anterior segment are obtained only if the whole ray energy of the mercury vapor lamp is used. Under these circumstances the ultraviolet rays, the ultrared rays and the whole visible spectrum are used. The result is necrosis of the cornea and of the iris, which naturally leads to changes of the lens in the pupillary region too. These experiments demonstrate the striking difference in the action of ultraviolet and ultrared rays. The former result in lesions of the corneal epithelium and in a transitory opacity of the aqueous; the latter damage the lens exceedingly. The widely accepted opinion that ultraviolet rays are very dangerous to the lens is wrong, as several authors were able to show by different methods.

P. C. KRONFELD.

Uvea

MELANOSIS OF THE IRIS AND NEW FORMATION OF A HYALINE MEMBRANE ON ITS SURFACE. D. J. WOOD, Brit. J. Ophth. **12**:140 (March) 1928.

The condition was seen in a woman, aged 63, who consulted the author for refraction. In the left eye the pupil was oval and eccentric upward. The iris, which was a dark grayish blue, bore on its surface in contact with the lens, on the nasal side only, a large pigmented plaque below. On biomicroscopic examination, at the lateral side the iris was raised up in a ridge which apparently reached the cornea. There were a few dark spots on the cornea below not like precipitates, and on the anterior capsule of the lens there was pigment suggestive of persistent pupillary membrane. Over the entire lateral half of the iris, from its root to the pupillary border, there were large numbers of raised brown nodes. At the upper border of the pupil, the uvea was everted and bore several nodes. The endothelium and the aqueous were normal. There was coronary cataract. Several months later, glaucoma was present. The nodes had spread all around the iris, and its upper sector bulged forward. The eye was enucleated sixteen months after the first visit.

According to E. Treacher Collins, who examined the specimen, the principal histologic manifestations were the presence of a layer of hyaline tissue, like that of Descemet's membrane on the surface of the iris; the abnormally deep pigmentation of a sector of the iris; the rounded nodules on the surface of the iris; the tion of a sector of the iris; the rounded nodules on the surface of the iris, and the at its junction with the ciliary body. Collins believes that the extension of the endothelium preceded the formation of the hyaline membrane, and that it is this endothelium and not that on the anterior surface of the iris which was responsible for its formation. The adhesion of the iris to the cornea is viewed as a congenital failure in separation of the iris from the cornea. As in the case reported by A. B. Reese, there is a dense pigmentation of the anterior layers of the iris over

the melanotic sector with gradations in the formation of clump cells, which supports the view that such cells originate in the chromatophores rather than that they are derived from the pigment epithelium. The nodes were mainly composed of endothelial cells and partly of a smaller variety of cells. It is suggested that they may be remnants of the iridopupillary membrane. Closely packed groups of unpigmented embryonal cells were found at the pupillary border of the iris, where it is adherent to the cornea and at its junction with the ciliary body.

The relation in which the several different features stand to one another is discussed. A general review seems to point to a primary congenital defect in connection with the iridopupillary membrane, resulting in its imperfect separation from the back of the cornea, so producing the anterior synechiae, and persistence of portions of it forming protrusions on the surface of the iris. There must also have been a formation of congenital nevus, resulting in the production of the abnormal area of pigmentation and areas of embryonic tissue. The endothelium of Descemet's membrane, due to the anterior synechiae, spread on to the surface of the iris and there formed an abnormal hyaline layer. This hyaline layer extending round the angle of the anterior chamber in a portion of its extent, as well as over the surface of the iris, must have caused some hindrance to the normal channels of exit for fluid from the eye, and so predisposed the eye to glaucoma. It is remarkable that symptoms of glaucoma did not appear earlier than they did. Possibly the areas of embryonic tissue were commencing to grow and manifest a malignant tendency, and this may have accounted for the recent onset of glaucomatous symptoms, which led to the excision of the eye.

W. ZENTMAYER.

THE TUBERCULOUS ETIOLOGY OF IRIDOCYCLITIS AND THE ETIOLOGY OF "IRIDOCYCLITIS WITH UNKNOWN CAUSE." A. V. FRISCH and A. PILLAT, Arch. f. Ophth. **121**:504 (Feb.) 1929.

The authors recommend the intracutaneous Mantoux test as sensitive and harmless in determining the tuberculous etiology of diseases of the eye. The intracutaneous reaction of 0.1 cc. of the three dilutions of old tuberculin (1 : 1,000,000; 1 : 100,000, and 1 : 10,000) shows the present allergic condition of the body better than any other skin test. At the same time it indicates the dose with which the treatment should be started. The subcutaneous injections of 0.2; 0.5; 1.0, and 5.0 mg. of old tuberculin often cause severe general and focal reactions which are avoided by using the intradermal method with much smaller doses. In a large percentage of cases with iridocyclitis, the ordinary medical and roentgen examinations do not reveal any pathologic condition. In most of these cases, the intradermal test indicates the tuberculous nature of the disease of the eye.

The authors state that most of the cases of chronic recurrent iridocyclitis are tuberculous, although the iris does not show any nodules. This opinion is based on (1) a careful and thorough medical examination which, in every case, was given by an internist who had the greatest experience in mild and mildest tuberculosis, (2) on the positive skin test and (3) in most cases, on the increased velocity of the sedimentation of the erythrocytes. In all of the cases described in this paper, the medical examination did not reveal any other pathologic condition than tuberculosis. In some of them, other ocular manifestations of tuberculosis were present (periphlebitis retinalis, retrobulbar neuritis and eczematous keratoconjunctivitis). The clinical picture of an acute diffuse iridocyclitis (iritis serosa Fuchs) can also be caused by tuberculosis. Usually the acute attack subsides after a few weeks and then the typical picture of chronic recurrent iridocyclitis

develops. In rare cases one attack of acute diffuse iridocyclitis is the only tuberculous manifestation that the patient shows and that heals without any sequelae. Many so-called rheumatic cases of iridocyclitis belong in this group of acute tuberculous uveitis.

The velocity of the sedimentation of the erythrocytes is increased in 50 per cent of the cases of nodular iritis. This indicates an active process with increased destruction of tissue. Cases of tuberculous iridocyclitis with normal velocity of sedimentation seem to have better prospects than the others.

Tuberculosis plays the most important rôle in the etiology of iridocyclitis, even more important than most European ophthalmologists believe.

P. C. KRONFELD.

Retina; Optic Nerve

THE RETINAL ARTERIAL PRESSURE IN THE POSITIONS OF SITTING AND LYING DOWN. SALVATI, Ann. d'ocul. **165**:917 (Dec.) 1928.

The author quotes Bailliart as stating in 1919 that the local arterial pressure was not influenced by lying down, the pressure remaining the same in the horizontal and the vertical positions. After calculating the difference between the level of the retina and the humerus, in 1920, Duverger said that the retinal arterial pressure was the same as the humoral.

Six patients were examined; the dynamometer of Bailliart was used, together with his technic, after much practice. The direct method of ophthalmoscopy was employed to determine the retinal pressure. Light pressure in the region of the external rectus muscle was found sufficient to cause minimum retinal pulsations.

In all cases the general blood pressure in the arm was taken, then the intraocular tension with the Schiötz tonometer, and finally, the retinal pressure when the patient was lying down and when he was sitting. The results of the determination of the retinal pressure were the same in two positions.

Francois Frank has shown that in the cerebral vessels there is no alteration in pressure following a change of position.

The retinal arterial pressure escapes the influence of change of position, while the systolic and diastolic pressures remain the same whether the patient is sitting or lying down. However, in persons with anemia and in those in whom the general mass of blood is reduced, there seems to be a local rise of pressure when the patient lies down.

H. W. SCARLETT.

Ocular Muscles

ABDUCENS PARESIS FOLLOWING SPINAL ANESTHESIA. A. NICOLATO, Arch. di ottal. **36**:27 (Jan.) 1929.

In forty-nine cases of abducens paresis following spinal anesthesia collected by Brunn in 1922, the third and fourth nerves were rarely involved. A man, whose Wassermann reaction was negative, was given 5 per cent procaine-epinephrine hydrochloride intraspinally for a gastric operation. Operative recovery was normal except for complete right abducens paralysis which developed after one week. The right twelfth nerve was also slightly paretic. The reflexes from the conjunctiva, nasal mucosa and external auditory canal were diminished but there was no disturbance of sensitivity nor any sign of meningeal irritation. Both labyrinths showed hyperexcitability to the galvanic and caloric tests. In most of the reported cases the paralysis occurred from five to twelve days after injection, which makes it unlikely that a hemorrhage in the nucleus of the sixth nerve could have occurred

as a result of the injection, as some authors have stated. The assumption of an arachnoiditis involving the nerve root would apply only to a few cases which have also shown signs of involvement of the cerebellopontile angle, or to those following injection of air or iodized oil for purposes of encephalography which cannot be compared to cases following simple spinal anesthesia. The theory of a toxic action of the drug on the nerve roots where they pass through the basal cisternae would explain the predilection for the sixth nerve, and the assumption that it is the split products of the drug which cause the paralysis would make the latent period more intelligible. The rarity of paralysis of this kind is difficult to understand, however, on any theory, and an individual predisposition may be necessary. The author states that his case could be classified as a toxic neuritis of the sixth and twelfth nerves.

S. R. GIFFORD.

Tumors

PAPILLOMA OR CANCROID? J. HEYMANS, Arch. d'opht. **46**: 48 (Jan.) 1929.

The author first reports the clinical notes and histologic description of a case of papilloma of the conjunctiva.

In these cases histologic examination definitely determines the diagnosis. The tumors, therefore, should be excised, as few of them penetrate the globe even when malignant; if malignant, surgical treatment can be given for recurrences.

The author points out the difficulty of correct clinical diagnosis, owing to the similarity of the two types of tumor not only in location but in configuration. For one type, only simple excision is necessary; for the other, eventual enucleation.

S. B. MARLOW.

Injuries

CATERPILLAR HAIR OPHTHALMITIS (OPHTHALMIA NORDOSA). R. FOSTER MOORE, Brit. J. Ophth. **13**:57 (Feb.) 1929.

A case is reported in a boy, aged 12. Fourteen days previously, the right eye had been struck by a thrown caterpillar, said to have been a "woolly bear." There was a general diffuse injection of all the ocular vessels; there was no staining of the cornea. The anterior chamber was half full of pus, and numerous fine deposits were present on the back of the cornea. The pupil dilated fully. Intolerance of light was a marked feature. The sight was greatly impaired. Three fine hairs were to be seen deeply embedded in the cornea. Attempts at removal failed. No hairs were to be seen in the conjunctiva, and no nodules were present at any time in the course of the disease. At the end of five weeks, the symptoms had largely abated. A considerable exudate in the vitreous had been mostly absorbed. The biomicroscope revealed several additional hairs extending deeply into the substantia propria. Three and a half months after the injury, the eye was quiet. The hairs could be seen apparently at their original site. Vision was 6/6. The exceptional features in the case were the early appearance of severe symptoms; the steady improvement, without exacerbations of the symptoms after obvious improvement set in; the absence of nodules in the conjunctiva episcleralis, and the complete recovery. It is pointed out that the barbs of the hair of the "woolly bear" caterpillar are directed forward toward the point of the hair and would therefore seem to be planned to prevent their penetration. While the suggestion that the hairs contain some form of poison appears to lack confirmation, it would seem that the severity of the inflammation with the formation

of hypopyon, and its long course, would strongly suggest that it was not due simply to the presence of a small, inert, foreign substance in the cornea, but that some potent poison was responsible for it.

W. ZENTMAYER.

Operations

A CASE OF MYASTHENIA GRAVIS OPERATED ON FOR PTOSIS BY HESS' METHOD. D. HARDIE, Brit. J. Ophth. **12**:31, 1928.

A man, aged 38, had had the disease for several years. The external muscles of the eyes and the levator on both sides were affected. The ptosis was bilateral. When the lids were raised, troublesome diplopia resulted. To avoid diplopia, only one eye was operated on. The operation was done by Kiep, who used the Hess method with excellent results. In the literature, the author could find reports of only three similar cases in which operation was performed.

W. ZENTMAYER.

THE MEASURED CORRECTION OF SQUINT BY COMBINED TECHNIC. W. H. LOWELL, Am. J. Ophth. **12**:173 (March) 1929.

The author speaks of the various disadvantages of the Worth operation and complete tenotomy, and describes his methods. In a case of convergent squint of 40 arc degrees the muscle is shortened 8 mm., as it is conceded that "5 degrees of arc" equal approximately 1 mm. of muscle shortening. The tucking is always performed first. The externus is exposed by a conjunctival flap, the convex portion of which is 2 or 3 mm. from the limbus, the legs extending out 1 cm. or more. The muscle is isolated, 3 mm. more than needed for the tuck. The hook of the Bishop tucker is passed under the muscle which is drawn up for 4 mm. A needle threaded with no. 4 twisted white silk is passed under the parallel bars of the tucker through the doubled muscle, 1.5 mm. from its edges, and a surgeon's knot is tied. A similar suture is passed through the other side of the muscle. These sutures are left to absorb. The wound is closed with two sutures; the upper includes the conjunctival flap, the upper corner of the tuck and the conjunctiva near the limbus. A similar suture is placed below. The internal rectus of the same eye is then similarly exposed and three alternate, transverse incisions are made, the middle three fourths through the muscle and the two end incisions a little over one half through it. The conjunctival flap is now sutured, and atropine is used. The dressing kept on for four days.

W. REESE.

Therapeutics

RAY TREATMENT OF CARCINOMA OF THE LIDS. S. DE VRIES, Klin. Monatsbl. f. Augenh. **82**: 145 (Feb.) 1929.

In a review of the development of the ray therapy in diseases of the eye, the author points out that more attention had been given to therapeutic results than to the prevention of incidental damage to the globe. Protection of the eyeball has been considered unnecessary by some authors because their experiments on animals led them to believe that cataracts are not caused by the rays. They overlooked the fact that the animals are killed shortly after the experiments, and that the cataract does not develop in the human eye prior to the lapse of a year or longer after the treatment. Filtrated rays are less damaging to the eye, but more protection is required.

Three points of importance must be observed in the treatment of cancer of the lids with rays, especially when radium is used: the prospect of a cure, the cosmetic result and the safety of the eye. These were adhered to in the treatment of forty-nine patients with carcinoma of the epidermis of the lids, consisting of chronic hard and indolent nodules or ulcerations which had resisted other therapeutic treatment; the glands were free in these cases. Microscopic examination confirmed the diagnosis in each case. The upper lid was affected in two cases only, the usual seat of the growth being the lower lid or the canthus, more frequently the nasal canthus. It was impossible to decide whether the growth originated at the lid or the margin. Conjunctivitis, trauma, warts, and, as in one case, a horny excrescence of the epidermis, were considered as possible origins. There was no evidence of an occupational origin. The area of the tumors was usually limited, involving the conjunctiva in four cases and the bone in one; in one case, it reached from one canthus to the other. Both sexes were equally represented. Twenty-five patients ranged in age between 50 and 70, fifteen between 70 and 80, and three between 80 and 90.

Combined roentgen-radium treatment was replaced by the radium therapy in 1920, and the radium was applied for twenty-four hours without interruption. So-called "fractional" application, in the author's opinion, is likely to produce a tolerance of the carcinoma cells to the rays.

Of these forty-nine patients, twelve were treated with roentgen rays only; four of them were cured directly, while three of the remaining eight could be healed with radium later on. The other thirty-seven patients were treated with radium. One of them received small doses without result, four had so-called "radium vaccination" with but one cured. The remaining thirty-two patients were cured with two exceptions; one of these required enucleation for panophthalmitis two months later, and the other had a recurrence six months later but did not return for treatment.

A recurrence was observed in another case five years after the treatment and was cured by a second exposure. The thirty patients who were cured were kept under observation for periods ranging from six months to eleven years. Comparing his results with those of Quigley who reported eighty-nine cures in ninety-six cases, and Regaud, who obtained thirty-three cures in forty-one patients, the author is convinced of the prompt action of the radium and of its lasting results.

The cosmetic results were most satisfactory. A smooth white spot or a few brown dots or telangiectasias indicated the former seat of the carcinoma. The palpebral margin appeared thinner in a few instances; the cilia were always missing in the treated area. Retracted scars or ectropium were never seen, except in one case in which the condition had existed prior to treatment. The conjunctiva usually showed hyperemia and some secretion for a few days only. Three patients had symptoms of a mild chronic conjunctivitis for several years. Four patients retained enlarged conjunctival vessels and one of them dilatation and contraction of the vessels as described by Birsch-Hirschfeld (*Strahlentherapy* **12:** 189, 1921). Fine epithelial lesions of the cornea, demonstrated by fluorescein, were noticed in three cases for a few months; they might have led to infections in the presence of purulent conditions of the conjunctiva or tear sac, as demonstrated by the case of panophthalmia. Preliminary treatment in these conditions is essential. Two cases showed localized atrophy of the iris and complete opacification of the lens which developed three and four years, respectively, after the ray treatment. Slight opacities of the lens, near the posterior capsule, were seen in the treated eyes of three patients with preservation of normal vision. The patients

had been treated five, eight and ten years previously. The vitreous and fundus remained free from changes. Chronic glaucoma developed in one case two years after a very strong exposure. Especially strong doses were given in those cases in which "fractional" treatment with radium or roentgen rays had previously been given.

The safest protection of the eyes against opacification of the lens is described by Den Hoed and Stoel who replaced the lead with other materials.

K. L. STOLL.

EXPERIMENTS ON THE USEFULNESS OF EYE PROTECTION IN RAY TREATMENT OF THE LIDS. D. DEN HOED, G. STOEL AND S. DE VRIES, Klin. Monatsbl. f. Augenh. **82**:158 (Feb.) 1929.

Photographic pictures are used to measure the intensity of the action of the radium on the various depths of the eye. This is done by placing small films between rubber, free from metal, at distances corresponding to those between the skin and the conjunctiva bulbi, the iris, the posterior pole of the lens and the retina, respectively. The radium is put on the surface of this "phantom." The darkness of the various films produced by the radium indicates its action on the different layers. The result is presented in the form of curves. Taking the surface dose as 100 per cent, 40 per cent penetrated the layer representing the conjunctiva, 20 per cent that of the iris, 10.5 per cent that of the posterior pole of the lens and 3 per cent that of the retina. The radium rays were filtered through 0.5 mm. each of silver and iron and 1 mm. of rubber.

Protection of the eye may be obtained in two ways: by insertion of a metal, like lead, in front of the eye, or by a larger distance of the radium from the eye and the insertion of a thick prothesis of wax. The first manner was considered inefficient, because the metal itself will emit α, β and γ rays, as shown in one of the curves. A lead foil, 1 mm. in thickness, covered with a thin layer of paraffin, will allow the conjunctiva to be reached by a larger dose of rays, namely, by 70 as against 40 per cent. Another curve showed the results after a lead foil of the same thickness, also covered with paraffin, had been placed between the conjunctiva bulbi and palpebrae; the conjunctiva of the lid received 77 per cent, as compared with 40 per cent through the paraffin alone. Even the skin received more during the use of the lead foil, due to its secondary rays. The intensity of the rays was nearly the same on the deeper layers with or without the lead foil. This is considered as proof that the conjunctiva bulbi and the iris are best protected by a greater distance of the rays, while the absorption of the lead is of consequence for the deeper layers.

A third curve demonstrates the results obtained through a lead foil, slightly over 2 mm. in thickness and covered on both sides with a thin layer of paraffin. The conjunctiva of the lid is reached by a trifle less and that of the globe by about one-third less than the rays received through the thinner lead plate, while the iris and lense are decidedly better protected. Admitting that the intensity of the rays is diminished by the lead foil itself, the authors think, nevertheless, that the main factor is the larger distance of the rays caused by the plate. This consideration, and the difficulty of placing so thick a plate into the conjunctival sac, prompt the authors to advise paraffin or another organic substance. A number of patients could tolerate a paraffin plate well after cocainization. The time of this tolerance is not mentioned. The use of an appliance of this kind is deemed necessary.

K. L. STOLL.

A New Prothesis for the Protection of the Eye Against Roentgen and Radium Rays. W. Rohrschneider, Klin. Monatsbl. f. Augenh. **82:** 162 (Feb.) 1929.

The recent number of publications on lesions of the eye caused by rays show the necessity for the protection of the eye. Experiments on animals, furthermore, performed by the author and others, demonstrated the sensitiveness of the lens to these rays. The conclusions drawn by Rados and Schinz from their experiments on animals, that the lens is one of the least sensitive organs to rays, are wrong. The globe can be well shielded only by a prothesis during ray treatments of the lids, the tear gland and the orbit. Mueller's lead glass prothesis, in use for a number of years, is not sufficient. Berthold and Grossmann showed that a lead glass prothesis would have to be 1 cm. thick to be useful. Hoffmann, therefore, uses a lead shell the inside of which is covered with shellac.

Rohrschneider constructed a prothesis of lead glass, the two walls of which include a space 2.5 mm. wide. This space is filled with mercury. The total thickness of his prothesis, including the walls, is from 3.5 to 4 mm. The mercury fills the space through a small tube at the convexity of the prothesis and the opening closed by melting. The weight of this appliance may be considered a disadvantage; yet it can be tolerated by the eye if inserted and removed after cocainization. A roentgenogram shows hardly any difference in the slight gray darkening of the film produced by either this prothesis or by a lead foil, 2 mm. in thickness, while the spot on which Mueller's lead glass prothesis has been placed appears dark gray.

K. L. Stoll.

General

A Case of Trichinosis with Exophthalmos. B. W. Key, Am. J. Ophth. **12:**178 (March) 1929.

L. S., aged 25, complained of tearing, headache and puffiness of the lids, and for the previous two or three days had felt weak and feverish and had had gastric disturbance.

Examination revealed edema of the face, especially around the eyes. The conjunctivae were chemotic, especially in the right eye and the lower fornices so that they protruded over the lower lids. The subconjunctival fluid was a striking yellow. The right eye showed marked proptosis and was directed down and a trifle in. The vision of each eye was 20/16; the corneas, irides and pupils were normal. The patient was hospitalized and found to have an eosinophilia of 69 per cent. A small section of the deltoid revealed *Trichinella spiralis*. A history of ingestion of raw pork four or five days prior to the illness was then obtained. The acute course of the disease lasted for seventeen days, and vision varied two or three times during that time. The fundi were negative throughout. A discussion of trichinosis and of different features of this case is included.

W. Reese.

Society Transactions

EDITED BY DR. GEORGE S. DERBY

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OPHTHALMOLOGY

Jan. 17, 1929

C. E. G. SHANNON, M.D., *Chairman*

EXTENSIVE BILATERAL RETINAL DETACHMENT IN ECLAMPSIA, WITH COMPLETE REATTACHMENT. DR. W. E. FRY (by invitation).

Dr. Fry reviewed the literature on this subject and reported two cases which occurred on the maternity service of the university hospital during a period of twenty-five months. During this time, there were 1,024 deliveries; among these there were 173 cases of late toxemia, an incidence of 16.9 per cent. Among the latter there were nineteen cases of eclampsia, an incidence of 10.9 per cent. This gave an incidence of 1.2 per cent for retinal detachment among the cases of late toxemia and of 10.4 per cent among the cases of eclampsia.

The first case was that of a colored woman, aged 36, who was admitted while in coma. There was bilateral retinal detachment, which extended forward 12 diopters in each eye. There were numerous exudates and hemorrhages. Complete reattachment occurred in three months, the vision was 6/9 in each eye and the visual field showed a slight concentric contraction.

The second case was that of a colored woman, aged 20, with marked edema of both lids and a chemosis. The retinal detachment was bilateral and extended forward 12 diopters. There were no exudates or hemorrhages. The detachment was still present twelve days later. The patient was not seen again for six months, when complete reattachment had taken place. The vision was 6/15 in the right eye, and 6/9 in the left. The visual field showed a contraction above in the right eye, but was normal in the left.

DISCUSSION

DR. W. ZENTMAYER: As I understand the Leber theory the fibrous tissue has its origin from the ciliary processes and courses parallel to the plane of the retina. If the detachment is due to such new-formed tissue, it is difficult to explain the reattachment. So far as the difficulty of explaining the possibility of the vitreous being dislocated by the elevated retina — except on the supposition of a hole in the retina — is concerned, it is to be recalled that one has solid detachment of the retina, which must give a similar condensation of the vitreous without the tears in the retina.

DR. FRANCIS HEED ADLER: Were any holes found in the detached retinas? Was the tension elevated immediately after the retinas had detached? Lister has pointed out the difference in prognosis between detachment with holes and those without holes, the latter being much more favorable, as would be expected.

Of the numerous theories of retinal detachment, the two most popular are the exudation theory and Leber's retraction theory. It would seem evident that these cases could be explained by the exudation theory, and the absence of holes would tend to support this view since the production of a detachment by means of bands

in the vitreous would almost necessitate the presence of tears which would allow fluid in the vitreous to come between the rod and cone layer and the pigment epithelium. One of the objections which has always been raised against the exudation theory is the supposition that one could not have such a detachment without either a rapid removal of fluid from the vitreous or a considerable rise of tension. In these cases, which seemed certainly due to exudation, if no rise of tension occurred it would prove that the argument was unsound.

TWO UNUSUAL EXPERIENCES WITH INFECTION BY THE STREPTOCOCCUS HEMOLYTICUS. DR. H. MAXWELL LANGDON.

This article will be published in full in the July issue of the ARCHIVES.

A NEW VISUAL TEST CARD. DR. ALFRED COWAN.

A test card arranged with letters was shown, which was previously described in the *American Journal of Ophthalmology* (**11**:625 [Aug.] 1928).

The letters are widely spaced so that each may stand out as an individual test object, and in order to eliminate fatigue as much as possible. The gradation follows closely the geometric progression proposed by Green, but the distances are so chosen that the Snellen fraction may be written in round numbers.

DISCUSSION

DR. ZENTMAYER: Dr. Cowan has given us a test chart based entirely on scientific principles, which, therefore, makes it ideal for recording visual acuity. We use test cards in our offices, however, not only for recording visual acuity but for the purpose of subjectively determining the refraction error, so that a test card constructed of letters which do not conform to a scientific basis for determining visual acuity are particularly suited for a determination of astigmatism. To resolve such letters as *"K"* and *"X"* requires that the astigmatism be accurately corrected, and such letters serve the purpose of deciding on the strength and axis of the cylinder, yet no worse letters could be constructed for estimating the visual acuity, because a one minute visual angle is lost as the angles of the letter are approached. It has always seemed to me that Landolt's optotype served well for estimating the visual acuity.

DR. A. COWAN: While this card was primarily designed as a step toward the standardization of test cards for the notation of visual acuity, the letters offer excellent objects for subjective refraction. At the limit of visual acuity the letters are easily modified; even a slight tilting of the card makes a noticeable difference in legibility. The Landolt *"C"* does not conform to the physiologic requirements of a visual test object. In this the patient is required only to locate the opening, and therefore, after a certain time, it becomes photometric and not a visual acuity test. The value of the visual acuity will always be too great with a test of this kind.

PANOPHTHALMITIS COMPLICATED BY PANSINUSITIS. DR. SYDNEY L. OLSHO (by invitation).

Miss C. J., aged 67, was first seen at the Jefferson Hospital on Sept. 28, 1927, complaining of poor vision. The right eye showed a leukoma of the cornea, the eye being quiet and the tension normal. The left eye showed numerous nebulae of the cornea, vitreous opacities and widespread myopic choroiditis. She disappeared to again reappear on Dec. 11, 1928, giving a history that the right eye

was painful and inflamed for eight days. The lids were red and swollen with ecchymosis of the conjunctiva. The globe was proptosed forward, downward and outward and was immobile. The roentgen rays showed a clouding of all the sinuses, and the rhinologist reported pansinusitis. A diagnosis of panophthalmitis was made. An evisceration was performed, following which pains subsided and there was a prompt and progressive disappearance of the edema and inflammation. Because of the rapid improvement following the evisceration, the panophthalmitis was thought to be caused by a perforating corneal ulcer and in no way due to an extension from the sinuses.

DISCUSSION

DR. W. ZENTMAYER: The question of the relative safety of enucleation and evisceration in panophthalmitis has been discussed for years. About twenty years ago, Dr. Randolph did some experimentation on animals hoping to reach a conclusion. My recollection is that the results were unsatisfactory. Although they leaned toward enucleation as a safe procedure, Dr. Randolph decided that personally he was not convinced, and preferred to treat the condition as he would an abscess.

DR. H. M. LANGDON: Would the result in this case have been different had an enucleation been performed?

The literature shows that meningitis is fully as frequent after the one as the other. Personally, I believe that there are other factors which determine the occurrence of meningitis in these cases. If there is not much orbital cellulitis I prefer to do an enucleation. When there is marked proptosis and swelling of the lids I do an evisceration.

A DAYLIGHT UNIT FOR PERIMETERS. DR. S. L. OLSHO.

Dr. Olsho showed a light unit that could be attached to the cylindric weight of a McHardy perimeter. It employs the same center of rotation as the arm of the perimeter and gives an even distribution of a daylight quality of light.

Dr. Olsho also showed a screen for perimetry. The screen consists of four hinged panels 6 feet (182.9 cm.) high and painted a neutral gray. This screen is especially valuable in a clinic for diseases of the eye, as it serves to isolate the patient, to exclude more or less from view objects distracting the attention and to provide a neutral background. It may be folded up flat or used as a cabinet to protect the perimeter.

DISCUSSION

DR. H. MAXWELL LANGDON: It strikes me that the concentration of the light in one rather bright area on the arm of the perimeter is not desirable if the field is taken in a room which is not generally well illuminated, as under such circumstances the retina would adapt itself for a low illumination and would be called on to do its work in a brightly illuminated field.

SUPERFICIAL NECROSIS OF CORNEAS CAUSED BY GAS EXPLOSION IN A HOT WATER HEATER. DR. BURTON CHANCE.

A man, while relighting the expired gas burner of a water heater, received the exploded blast of air filled with dust particles on the exposed corneas. When examined a few minutes later, the surfaces of the corneas were grayish and lacked luster, like the eyes of the dead. In the course of the next few days, the entire epithelium exfoliated, but by the end of the eighth day it was completely reformed without further consequences to the laminas and to the sight.

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

THOMAS H. JOHNSON, M.D., *Secretary*

Jan. 21, 1929

BERNARD SAMUELS, M.D., *Chairman*

MUCOCELE OF THE POSTERIOR ETHMOID SINUS, WITH ORBITAL SYMPTOMS. DR. J. W. BABCOCK.

C. H., aged 41, had been under observation at intervals for six years for occasional nasal obstruction due to deviation of the septum and acute sinusitis, but these conditions were never troublesome enough to warrant operation.

The illness present at the time of presentation began in March, 1928, when the right eye became prominent, and there was some pain in the right orbit following extensive use of the eyes. Dr. Babcock first saw the patient in May, 1928, at which time he irrigated the right sphenoid, finding it clear, with a membrane slightly thickened to the touch, and with a small mass of adenoids at the vault. From these observations, he believed that tumor of the orbit was the probable diagnosis

Twelve days later he saw the patient again, at the request of Dr. Arnold Knapp. The patient's right antrum was slightly dark to transillumination, and showed a small mass of mucopus on irrigation. With repeated irrigations, the patient said he felt better, subjectively, but there was no change in the proptosis. Radiograms taken by Dr. Imboden showed a slight clouding of the right antrum and a peculiar absorption of the inner wall of the right orbit. Dr. C. G. Coakley also saw the case at this time, and both felt that the sinusitis was coincidental, and not responsible for the orbital tumor. However, urged by Dr. Knapp, as a less formidable procedure than exploration of the orbit and one likely to disclose the true nature of the disease, on June 25, 1928, Dr. Babcock removed a portion of the right middle turbinate in order to explore the area. Expecting to find a malignant growth of the ethmoid, he was surprised at almost the first forceps bite into the anterior ethmoid region to open a cavity running backward to a depth of about 1½ inches (3.77 cm.), containing a yellow oily fluid resembling Hollandaise sauce, which was under no tension. Immediately, the patient said he had diplopia for the first time. After seeing that the cavity was widely opened and emptied, no more was done. The opening had remained patent in spite of an acute inflammation involving the right antrum and ear, and the diplopia and exophthalmos had disappeared, not to return. Five days after the opening of the cyst, the patient had an acute conjunctivitis, which subsided promptly.

The character of the cyst was unique to Dr. Babcock, and he suggested an inclusion cyst or a dermoid, which, however, he had never seen in this location. It did not seem to him to be a mucocele. Although the patient had had pulmonary tuberculosis, which was supposed to have been arrested years before, there seemed to be no relation between that condition and the condition present at the time of presentation.

DISCUSSION

DR. ARNOLD KNAPP: The case presents a number of interesting features, which are presented in Dr. Babcock's report (to be published in full). The eye symptoms suggest a process near the apex of the orbit. With clouding of the ethmoid, and a blurring of the dividing line, it does not seem that an orbital

process would produce these changes in the ethmoid. On the other hand, I believe that the eye symptoms are not active enough for a primary orbital process, and that the burden is therefore thrown on the ethmoid. An interesting paper was read in 1920, in which Dr. van der Hoeve described a case of mucocele of the sphenoid and posterior ethmoid cells with optic atrophy (*Nederl. Tijdschr. v. Geneesk.* **1**:1431 [April 24] 1920; *Ztschr. f. Augenh.*, 1920).

The contents of the cyst seem to indicate mucocele. Mucocele contents vary from clear to chocolate-thick material, always containing a great many cholesterol crystals.

DR. J. W. BABCOCK: I had the impression that mucoceles contain a sticky mucoid material. The fluid from the cyst described was oily, with almost a metallic glitter with marked yellowness. I could easily imagine that a change from columnar epithelium to squamous epithelium might alter the contents of the cyst.

DR. LEWIS F. CRIGLER: Might not the cyst be of a hematomatous nature? About a year ago, I saw a man who had sustained a blow over the left brow in an automobile accident. For six months there was no deformity save for slight swelling and drooping of the upper lid, and then the eye began to protrude. Roentgen studies showed complete absorption of the supra-orbital wall, supporting a clinical diagnosis of orbital tumor; on the basis of this observation an operation was advised. A horizontal incision through the skin of the left brow and dissection exposed an encapsulated mass which filled the frontal sinus and extended into the orbit above and behind the eyeball. On rupture of the wall of the cyst, there was encountered a cheesy, chocolate-colored material which gave rise to the assumption that a blood clot had undergone cystic degeneration. The contents of the sac and the entire frontal sinus were obliterated. Recovery was uneventful except for a persistent diplopia accounted for by interference with the pulley of the superior oblique muscle. The exophthalmos completely disappeared.

DR. ARNOLD KNAPP: Is it not possible in Dr. Crigler's case that the blow might have brought to light the cyst that had been present in a latent condition before the blow was received?

DR. J. W. BABCOCK: The cyst I described did not resemble any hematoma that I have ever seen.

METASTATIC CARCINOMA OF THE CILIARY BODY. REPORT OF A CASE.

This article was published in the May issue of the ARCHIVES, p. 604.

CAVERNOUS ANGIOMA OF THE ORBIT CAUSING PRESSURE SYMPTOMS ON THE EYEBALL. DR. ARNOLD KNAPP.

This article appears in this issue, p. 704.

DISCUSSION

DR. MARTIN COHEN: Did a hemorrhage occur during the operation in the second case of Dr. Knapp? What did the microscopic examination show? Could this growth be explained as being due to fetal displacement?

DR. ARNOLD KNAPP: No hemorrhage occurred at operation. The mass was encapsulated and was shelled out easily, and the microscope showed that it belonged to the cavernous angioma group.

COMPLETE PARALYSIS EXTERNAL RECTUS: TRANSPLANTATION OF TENDONS OF VERTICAL RECTI. DR. BEN WITT KEY.

This paper will be published in full in the July issue of the ARCHIVES.

DISCUSSION

DR. JAMES WATSON WHITE: In my opinion, it is almost impossible to discuss such a case, unless one can study it before operation. Sometimes in these cases of congenital external rectus paralysis, the secondary contracture of the internus is so great that there seems to be no abducting power. When the internus is tenotomized, however, one does find some action of the externus. That is a possibility in the case presented. Whether the result in this case is due to a too free tenotomy of the internus with some abducting power of the externus, or to the transplantation can never be proved. I have never done the transplantation myself, because I cannot convince myself that it is the thing to do.

DR. CONRAD BERENS: I performed this operation on a patient who had congenital abduction deficiency with retraction. I should like to know if in this case any benefit could be obtained from transplantation of the temporal halves of the superior and inferior recti, without disturbance of the internal or external rectus. No improvement in abducting power, or increase in external rotation, could be detected.

DR. BEN WITT KEY: The question of function in this case is interesting. I did not believe it was possible to secure any motion outward; however, I hoped to bring the eye to the median line for the cosmetic result. In spite of this, I did secure an outward motility of 10 degrees; the oblique muscles then came into play as supplementary abductors. I cannot explain this function as being due to the transplanted recti.

DR. E. F. KRUG: Was notice taken of the position of the eye on which operation was performed compared with that of the other eye, when the patient became anesthetized?

DR. BEN WITT KEY: There was no motion of the eye outward before the patient was anesthetized, and no change was noted during the time the patient was under anesthesia. Since the eye was apparently fixed, I think that any change in its position would have been noticed. When tenotomized, the internal rectus did not indicate a muscle which was in traction to the degree that it would relax at all to an opposing action; I thought this evident, considering that there is a convergence of 20 degrees now.

DR. RUDOLF AEBLI: It seems to me that four steps were performed in this operation at the same time; namely, the tenotomy of the internal rectus, the resection of the external rectus and the transplantation of the two muscle bellies of the superior and the inferior recti. I think that it is difficult to say which one of the four steps produced the result. Only by doing the operation in steps could one be in a position to state that the moderate abduction obtained was the result of the transplantation of vertical recti tendons.

DR. BEN WITT KEY: On account of motility inward, I believe that the internal rectus was more than a fibrous band. With complete paralysis of the externus, one would expect little or no result unless the internus was tenotomized. In this case, tenotomy of the internus was done to give as much effect as possible by the attachment of the vertical recti tendons. In reply to Dr. White's statement, I believe that the convergence was not due merely to a fibrous band holding the eye fixed inward, because there is now a definite motility of about 20 degrees inward.

POSTOPERATIVE TRAUMATIC PULSATING EXOPHTHALMOS. DR. MARTIN COHEN.

The patient was presented before the section at the November meeting. As conservative treatment had no effect on the exophthalmos or on the cephalic

bruit; both of which resulted from a basal fracture, the patient was operated on by Dr. G. M. Phelps. The operation consisted of the ligation of the internal carotid artery, with the patient under local anesthesia, using 2 per cent procaine hydrochloride solution. A skin incision, 2 inches (5 cm.) long, was made along the anterior border of the sternocleidomastoid muscle, the center of the incision being on a level with the middle of the thyroid cartilage. After exposure, the internal carotid artery was ligated just above the bifurcation from the common carotid. In order to effect a complete closure, two ligatures were placed, and the space between the two ligatures was then crushed with an artery clamp. After the first ligature was applied, the cephalic bruit ceased. The wound was closed with silk sutures, and the patient made an uneventful recovery, leaving the hospital in two weeks. The exophthalmos, however, remained stationary for six days, after which it suddenly disappeared, and it had not recurred at the time of presentation. When the patient left the hospital, the vision was 20/20, and the field and tension were normal. There was still a slight limitation of motion of the eyeball outward, and upward and outward. There was also a partial ophthalmoplegia interna. These complications are probably of mechanical origin, owing to the pressure of the enormously dilated orbital ophthalmic veins and their branches, and not due to any direct traumatic involvement on the cranial nerves in the cavernous sinus resulting from the basal fracture or an early thrombus. The fundus was normal.

ORBITAL CAVERNOUS HEMIANGIOMA. DR. MARTIN COHEN.

The patient was presented before the November meeting of the Section as having an orbital growth of unknown origin. The mass was first noted sixteen years before presentation, then remained comparatively stationary for about ten years. She was admitted for operation on Nov. 26, 1928. With the patient under general anesthesia, through a conjunctival incision extending from canthus to canthus along the upper retrotarsal fold, a firm nodular bluish mass was exposed, lying within the cone of muscles. With blunt dissection, the mass was separated from the surrounding structures, and it was then severed with scissors near the apex of the orbit, sacrificing the optic nerve. Slight hemorrhage was encountered, but readily controlled. A corneal ulcer developed, growing into panophthalmitis, which required evisceration of the globe. The orbital growth was 5 cm. in diameter, and of firm consistency. Tissue section showed many independent blood spaces, all containing red and white blood corpuscles and lined with endothelium, rendering a diagnosis of orbital cavernous hemangioma.

CIRCUMCORNEAL TRANSPLANTATION OF BUCCAL MUCOUS MEMBRANE AS A CURATIVE MEASURE IN DISEASES OF THE EYE. DR. RUDOLPH DENIG.

This paper was published in full in the March issue of the ARCHIVES, p. 351.

DISCUSSION

DR. THOMAS HAYES CURTIN: I have obtained success from grafts in eczematous cases, provided the graft is not done while active inflammation is still in progress in the eye. In acute stages of trachoma or eczema, peritomy is better than a graft.

DR. ERNEST F. KRUG: In the case of lime burn rendering the cornea entirely opaque, and the vision barely light perception, I applied a graft within two weeks after the accident, and now the cornea is beginning to clear, and the patient can count fingers at 5 feet (152.4 cm.).

DR. WENDELL I. HUGHES: I should like to know if Dr. Denig employed his operation in xerosis.

DR. ARNOLD KNAPP: In what way does the graft exercise its benefit in cases of burns of the cornea?

DR. DAVID H. WEBSTER: What suture material does Dr. Denig use?

DR. LEWIS W. CRIGLER: How often was it necessary to remove the flap and replace it with conjunctiva for cosmetic result?

DR. ERNST WALDSTEIN: I saw a patient with a case of stubborn dystrophy of the cornea who obtained no relief from six months' treatment here and abroad. The condition disappeared after Dr. Denig performed his operation.

DR. M. A. SCHWAGER: Would one be justified in trying a graft in any corneal opacity, irrespective of its origin?

DR. FRANCIS W. SHINE: Would Dr. Denig do a complete circular graft, or only a partial graft, in a case of lime burn of the cornea?

DR. RUDOLF DENIG: Transplantation should be done for opacities attending inflammation of the cornea which originates in the limbus. In sclerokeratitis, transplantation has been found superior to tuberculin injections or to roentgen therapy. Favorable results may be expected in acne rosacea and in herpes lesions. I have never operated for xerosis. In answer to Dr. Knapp's question, I believe that burned and chemically decomposed conjunctiva should be removed and replaced by healthy mucosa from the mouth, and I attribute the benefit to an increase of nutritive material filtering from the limbus into the cornea. The graft should be placed opposite the burned area before epithelization sets in. In a number of patients, I have removed the graft for cosmetic reasons at the patient's request. As a rule, the graft grows paler in time, and the patient does not care to have it removed. For sutures, I use silk in the conjunctiva and catgut in the mouth. I have never seen any complication from wounds in the mouth.

FITTING OF ARITIFICIAL EYES, WITH ESPECIAL REFERENCE TO GOLD BALL IMPLANTATION. MR. PIERRE GOUGELMANN.

This article will be published in full in the July issue of the ARCHIVES.

MUCOUS MEMBRANE GRAFT FROM THE MOUTH TO THE EYE. DR. ERNEST KRUG.

The patient was first observed in August, 1927, showing an infection superimposed on a trachomatous conjunctiva. All four lids were thickened, and each cornea was covered with pannus, the vessels coming in from the entire circumference. Each recurrent attack showed vesical formation on the eyelids and on the cornea, suggesting the possibility of pemphigus. The serum from these vesicles was found on culture to be sterile. All constitutional tests were negative. The vision of the right eye was never better than 15/100, and that of the left eye, 10/200. Denig's graft operation was done on the right eye on Jan. 4, 1928. Slowly, the right cornea had cleared, allowing a vision of 20/30 with a correcting lens. The vision of the eye on which operation was not performed remained 15/200.

GUTTATE KERATITIS. DR. H. D. SMITH.

A Russian woman, of Jewish extraction, aged 45, married, with four children, was presented. Her grandmother was blind at the time of her death. Her mother, now 67 years old, at 55 began to lose her sight. She had a brother, aged 43, and

a sister, aged 39, with the right eyes affected; a brother, aged 32, had both eyes affected. Three remaining brothers, aged 38, 36 and 34, had unaffected eyes. The patient was overweight, and she had had an operation for gallstones in 1918, and a hysterectomy, in 1925, for a fibroid tumor. Two years before presentation, there was a complete loss of the sense of smell from no apparent cause. On physical examination, the chest and abdomen were normal. Chemical analysis of the blood showed: dextrose, 99; nonprotein nitrogen, 30, and urea, 15 per hundred cubic centimeters. She gave no history of tuberculosis, but reacted positively to the Pirquet and Mantoux tests.

Fourteen years before presentation, she noticed a scratchy sensation of the right eye, and since then the condition would flare up three or four times each year, remaining fairly painful for one or two weeks. The vision in the right eye had become progressively worse, until at the time of presentation only hand movements could be perceived. About two years previously the left eye began to have similar attacks, the vision as presented being 20/30. The fields were normal in each eye. The right cornea was anesthetic, the left normal. There was grill-like degeneration of the stroma of the cornea, the opacities having no termination as one would expect of nerve fibers or blood vessels. This condition was described by Biber and Haab as "gittrige keratitis"; by Groenow and Fuch as "nodular or lattice opacities of the cornea" and by Fehr as "family punctate degeneration of the cornea." The most comprehensive description was by Gallemaerts, "examen microscopique des affections de la cornea." No definite theory had been advanced as to the underlying pathologic process and treatment was of no avail.

DISCUSSION

DR. ARNOLD KNAPP: Has Dr. Smith observed the similarity of the process in the mother and that in the brother? I have never seen a lattice keratitis which showed the extensive change that the condition of this patient showed.

DR. H. D. SMITH: I have not seen the other members in the family.

CAPSULOTOMY SCISSORS. DR. SAMUEL P. OAST.

The capsulotomy scissors exhibited by Dr. Oast was designed for control by thumb and forefinger, and to this end it is short and light, with particular attention to the sensitiveness of the spring. The instrument may be operated through a 3 or 4 mm. section made preferably by an Agnew keratome.

NEEDLE HOLDER. DR. SAMUEL P. OAST.

A short, powerful needle holder without catch was shown by Dr. Oast. It required slight pressure of the thumb and forefinger, and was particularly adapted to conjunctival suturing.

TUMOR OF THE ORBIT. DR. THOMAS H. CURTIN.

A man, aged 70, was first seen in March, 1928, on account of edema of the upper lid and ocular conjunctiva of the right eye, which subsided under treatment with zinc drops. The same condition recurred in January, 1929, without proptosis or limitation of movement of the affected eye. Digital examination revealed a small mass above the globe and free from it, entirely painless to pressure. X-ray pictures showed no evidence of tumor, but an absence of the frontal sinuses and a clouding of the left antrum. Dr. Curtin raised the question whether or not a biopsy should be attempted for diagnosis, or whether the mass should be removed entirely.

DISCUSSION

DR. MARTIN COHEN: It is difficult to make a diagnosis of orbital growths in men of that age. I have seen improvement in cases of this kind under stringent antisyphilitic treatment. If antisyphilitic treatment is of no avail, an operation should be done. I should advise the patient to have exenteration done, if frozen section at operation should reveal osteosarcoma.

Regular Meeting, Feb. 18, 1929

THOMAS H. JOHNSON, M.D., *Secretary*

BERNARD SAMUELS, M.D., *Chairman*

A CASE OF SYMPATHETIC OPHTHALMIA OCCURRING FORTY-EIGHT YEARS AFTER INJURY, WITH MICROSCOPIC SECTIONS. DR. JAMES W. SMITH.

This paper will be published in full in a later issue.

DISCUSSION

DR. MARTIN COHEN: I have had the impression for some time that the insertion of a glass ball or a gold ball into Tenon's space in cases of traumatic iridocyclitis may set up a sympathetic ophthalmia. I believe there should be a word of caution said about the insertion of this foreign material, as it is apt to cause pressure symptoms on the stump of the optic nerve and the surrounding vascular tissue. Perforating injuries resulting in traumatic iridocyclitis are better treated in the usual way. The cosmetic result gained by the insertion of foreign materials is not sufficient to warrant any risk.

THE VALUE OF TUBERCULIN IN OPHTHALMOLOGY. DR. CLARENCE KING.

This article appears in full in this issue of the ARCHIVES, p. 713.

DISCUSSION

DR. GEORGE S. DERBY: Dr. King has given a thoughtful, clear and restrained paper on the use of tuberculin. Like perhaps the majority of ophthalmologists he is an enthusiastic believer in its efficacy. He cites some valuable cases in which he believes that tuberculin was of great benefit. He is kind enough to refer in a complimentary way to a paper on anterior ocular tuberculosis which Dr. Carvill and I presented at the meeting of the American Ophthalmological Society in 1927. I am going to base my remarks chiefly on that paper as it summarized to a large degree the observations we had been making at the Massachusetts Eye and Ear Infirmary during a period of about twenty years. The average length of time the cases were observed was fourteen years. Dr. Carvill and I felt, and still feel, that as tuberculosis was a chronic disease and subject to periods, more or less long, of quiescence, often only to light up again in fresh attacks, it was incorrect to speak of a cure. Any one who has ever had tuberculosis, and almost every one has had it, has a focus lying quiescent in the body. Whether this focus is going to light up and become active and other foci appear depends on a large variety of circumstances. Under conditions of lowered resistance which may occur in every one, the tuberculous focus lights up and one has a fresh attack. Thus, unless a patient has been observed over a long period, one cannot

really know much about his disease or form a correct idea of what one's treatment has accomplished. Frankly speaking and with due deference to my friend Dr. King, and to the other adherents of tuberculin therapy, we do not believe that most of the articles written about the use of tuberculin tell the whole story, because they are based on series of cases which have been observed over much too short periods. We said in our article that "like tuberculosis of the lung, anterior ocular tuberculosis may be arrested but it is incorrect to speak of a cure." We confined our attention to anterior tuberculosis because we wished to give observations only on cases observed over the longest time in our power, and when we started our work many cases which would now be recognized as tubercle of the iris, ciliary body, choroid and retina could not be recognized as they can today. On the whole, I believe that what we said about tuberculosis of the anterior membranes of the eye can just as well be applied to most forms of ocular tuberculosis, although I think that the posterior forms are, on the whole, more resistant to treatment and less likely to heal spontaneously. To quote again, "until large series of cases shall have been followed throughout life we shall not know the truth about ocular tuberculosis." Now there are comparatively few series of cases in the literature which have been observed a long enough time to make the observations of real value. Most of the literature on tuberculin is based on the behavior of the eye in one attack. No cognizance is taken of the frequency of recurrence. Tuberculosis of the eye like tuberculosis elsewhere is characterized by its chronicity and tendency to recur. While under observation, in all but seven of our sixty-three patients there were recurrences: ten had 2, twelve had 3, five had 4, four had 6, six had 7, one had 8, two had 9, and the remaining patients had, respectively, 10, 11, 12, 13, 14, 15 and 16 recurrences. In seven patients no recurrences were observed, fifty-six had 304 recurrences, between 5 and 6 per patient. Many of our patients remained free from recurrence over a long period. We have data of sixty-nine quiescent periods ranging from three to eighteen years. Six of our sixty-three patients eventually died of phthisis, a high mortality rate, more remarkable because none of these patients had active pulmonary lesions when they came to us, some of them had been treated at sanatoriums for their ocular tuberculosis and all of them had the best advice regarding general hygiene that could be given to them by our internist and the cases were intensively followed up by our social worker.

Our experience with tuberculin has been considerable but we have not used Toniessen's tuberculin. In a few instances we believe that the process has been favorably influenced. We believe that the injudicious use of tuberculin in two cases led to blindness. Of course this might happen with any strong drug if given injudiciously and should not be held up against tuberculin. In a number of cases we have felt that tuberculin has been harmful. In the vast majority of cases there has been no effect. It seems possible that if a dose could be administered in favorable cases which would stir up only a mild reaction, the patient might be benefited. If one falls short of this point no result is to be expected; if one oversteps it harm is likely to be done. I do not believe that the most skilful man in the world can accurately gage such a dose; there is entirely too much guess work in tuberculin therapy. Many patients tend toward spontaneous recovery; they are given innocuous doses of tuberculin and tuberculin gets the credit for their recovery. Six months, two years or five years later they have a recurrence, but they have passed out of the care of the man who attended them before and they are recorded as cured. Every year sees new preparations of tuberculin as well as other methods of treating this very serious disease, which would seem to indicate at least a certain dissatisfaction with present methods.

Some use one preparation, some another, some use minute doses and some insist on producing reactions. Some say the indications for the use of tuberculin are clear, others that they can be arrived at only after careful observation. Some say that it should not be used in the exudative forms of the disease. Loewenstein says he sees little difference in the healing tendency in exudative and nonexudative processes. Presumably most of the writers on tuberculin give their patients the benefit of the standard treatment for tuberculosis, rest, fresh air, general hygiene, proper diet, heliotherapy and the removal of foci of infection. It may be said, however, that this treatment is passed over in a few words and pages are devoted to the benefits of tuberculin. Is it any wonder then that men of little experience are apt to get a false estimate of the value of the drug and lay too little stress on the only methods which have withstood the test of time in the treatment for tuberculosis?

Tuberculosis of the eye, like pulmonary tuberculosis, is a chronic and recurring disease which may be overcome for a time and held in abeyance but is extremely likely to recur if the resistance becomes lowered. The fact that our sixty-three cases showed a mortality rate of nearly 10 per cent from phthisis should give rise to thoughtful consideration. These patients were of the hospital type, had to earn their own living and thus were more prone to have a lowered resistance. It may be said, however, that, while in our tuberculosis class, the patients were carefully instructed in hygiene and checked up by means of a personal book in which were recorded the temperature, the pulse rate, the number of hours spent in the fresh air, the sleep and the diet. Their home conditions were carefully supervised, and when the disease was particularly resistant treatment in a sanatorium was advised and carried out in fourteen of the sixty-three cases. Few hospitals for diseases of the eye are equipped to give their patients adequate antituberculosis treatment, nor is it always easy to do so in private practice. In such instances it is probably wise to send the patients to a sanatorium for a number of months, not only for the treatment they get during that time but for the knowledge they obtain of the disease, and the means to combat it, for the contest against tuberculosis must be regarded as a lifelong fight and not a contest of a single round.

The average attack of ocular tuberculosis is usually self-limited under proper hygiene. The process will clear up under tuberculin therapy if used with judgment, and also without tuberculin. A slight attack may clear up in a few weeks, a severe attack may last well over a year. The disease then becomes quiescent, and sooner or later another attack is to be feared. There is little proof that the continued use of tuberculin after the attack is over will prevent recurrences, although some writers state that this is the case. Others express a contrary view. The main conclusions which, in my opinion, may be drawn are as follows:

Observations on ocular tuberculosis have only a limited value unless the cases have been followed over a long period.

Tuberculosis of the eye like tuberculosis elsewhere is often characterized by recurrence.

The object of treatment is not only to overcome the present attack but, as far as possible, to prevent recurrence. Moreover, it should be remembered that tuberculosis may show itself in other parts of the body, and pulmonary tuberculosis may appear in these cases and lead to a fatal issue.

The mainstay in the treatment for all forms of tuberculosis consists in measures to build up the general resistance of the patient, fresh air, rest, proper diet, heliotherapy, long continued treatment and observation. In our enthusiasm for a specific form of therapy let us not forget the foundation stones in the treatment for this disease.

DR. JOHN WEEKS: As many of those present know, I have been an interested observer for many years of the effect of tuberculins in ocular tuberculosis. In a paper read before the Colorado Congress of Ophthalmology and Oto-Laryngology in Denver, August, 1925 (*Am. J. Ophth.* **9**:243 [April] 1926), the following appears: "In 1910 the writer reported 56 cases of tuberculosis of the eye treated by the use of tuberculins, in which there were 35 recoveries and improvement in 15 cases. In one case removal of the globe was necessary. Since 1910 the number of cases treated has made a total of 100 at least." A few additional cases have been observed since 1925. The treatment of the greater number of the patients has been under my personal supervision. In my opinion, the results since 1910 have been as good as they were before that date.

Since, as we all know, ocular tuberculosis, with but few exceptions, is a metastatic manifestation of the disease, it is desirable to determine the site of the tuberculous process in other parts of the body. To this end a thorough physical examination should be made; a roentgenogram of the chest should be taken and the plates competently interpreted. All other possible sources for the pathologic process should, of course, be investigated. It should not be forgotten that syphilis may accompany tuberculosis, as well as other systemic diseases. The study of the case should include an examination as to the presence of this disease.

The use of tuberculins in establishing a diagnosis of ocular tuberculosis is indispensable in most cases. In sclerosing keratitis, in episcleritis and in some forms of iridocyclitis a positive diagnosis is virtually impossible without the use of tuberculin. When the proper diagnostic dose of tuberculin is given and a focal reaction presents — the focal reaction is essential — one is positive that the pathologic process is tuberculous. I should not say that tuberculin will produce a specific reaction in every case of tuberculosis affecting the eye, but the percentage of failures is certainly small.

In applying the diagnostic test in cases of tuberculosis of the eye I have obtained the best results by the use of "old tuberculin." Other tuberculins have been used but were not so satisfactory. The reaction to the intradermal and Calmette tests is purely local. Unless a considerable amount of tuberculin is employed, the reaction in the von Pirquet test is also only local. I have seen two cases in which the application of the von Pirquet test was followed by a decided systemic and focal reaction, this because of the introduction of a rather large quantity of tuberculin into the circulation. These tests are valuable in determining the presence of tuberculosis in some part of the system but not as a diagnosis for small, localized, tuberculous lesions. (The method of the diagnostic use of tuberculin that I have advocated may be found in the article previously referred to, also in my treatise "Diseases of the Eye," Philadelphia, Lea & Febiger, 1910.)

In regard to the use of tuberculins for therapeutic purposes, a favorable result often depends much on the ability of the surgeon to manage properly the treatment of the case — to increase or diminish the dose — to regulate properly the time of giving the dose and to control properly the habits of the patient. To do these things well implies a constant, accurate knowledge of how the case is progressing. The surgeon should be aware of the fact that preparations of tuberculin are sometimes inert and he should be prepared to change to another if the preparation in use does not produce the desired effect.

In the presence of active, extensive tuberculosis in other parts of the body the use of tuberculins is contraindicated. Recurrences in ocular tuberculosis, even after great precaution in the treatment with tuberculins and after a stage of quiescence of some months' duration has been reached, are not uncommon. They are usually accompanied by a depression in the general physical condition of the

patient. A resumption of the treatment with tuberculins usually results in a subsidence of the active process. In my experience recurrence in tuberculosis of the eyeball is much more common than in tuberculosis of the ocular adnexa. Recurrence in corneal tuberculosis has been nearly as frequent as in tuberculosis of the uveal tract. In my opinion it is desirable to continue the treatment with tuberculins in all cases of ocular tuberculosis, perhaps in slightly reduced dosage, for at least three months after the tuberculous process has become quiescent, in the endeavor to prevent recurrences.

As a result of my experience in the use of tuberculins in the treatment for ocular tuberculosis, I wish to state that I am fully in accord with the conclusions arrived at by Dr. King. In the face of all that has been stated to the contrary and with the knowledge that spontaneous recovery from tuberculous diseases of the eye sometimes occurs, I believe that in tuberculins one has valuable agents for the diagnosis and treatment of ocular tuberculosis and that the ophthalmic surgeon who fails to use tuberculins in the treatment for such cases fails in his duty to his patient.

DR. A. B. FERGUSON: Dr. Samuels is right in saying that I know nothing about ocular tuberculosis, but Dr. Samuels and Dr. Knapp were insistent that you would be interested in the observations of the roentgen examination of the chest in cases of tuberculosis elsewhere than the chest, and recently I have seen, through the courtesy of Dr. Knapp, roentgenograms of the chests in seven cases of ocular tuberculosis, and have also seen a great number of roentgenograms of the chest in cases of tuberculosis of the bones and joints; it is on my experience in the latter type of case that I shall base the remarks that I have to make.

The changes found in the lungs are not necessarily those of active tuberculosis with which one is apt to be more familiar. Of course, there may be an active pulmonary tuberculosis occasionally in these cases of tuberculosis elsewhere, but for the most part what one finds in the chest is the residual of a previous pulmonary infection, and the classification of that residual is frequently difficult and in some ways confusing. The few remarks that I wish to make are concerned entirely with the classification of the sclerotic changes with reference to their significance as indicating a previous tuberculosis. There are many diseases of the lung that may leave sclerotic changes, that is, the formation of scar tissue to a certain degree in the lymph nodes or along the bronchi or in infiltrations in the pulmonary tissue. Any disease which will produce sclerosis will leave its mark so that one has to differentiate between the sclerosis left by nontuberculous processes and the sclerotic changes that are left by tuberculosis itself. First I must describe, so far as I can, how sclerosis is evidenced in the roentgenogram and that is a rather difficult thing to describe because, to a large degree, whether one concludes that sclerosis is present or not will depend on one's judgment of the variation of density, for example, with a certain degree of density in a given shadow one would conclude that it was sclerosis, whereas with a somewhat less degree one would conclude that there was less sclerosis. Such a matter cannot be demonstrated, but is merely a question of judgment; hence it is a matter in which one observer may differ considerably from another, and it is almost impossible to describe it in words. This condition cannot be produced on lantern slide projections with accuracy. I have brought some original films along that will be available for you to view at close range when the opportunity arises. Sclerosis is evidenced by an increase in density in the parts that become sclerotic — the lymph nodes, the bronchial trees or the pulmonary infiltrations — and that increase of density is accompanied by sharpness of outline. Other types of infiltration would produce an increase of density, but instead of keeping the outlines of the trunks

sharp, the infiltration fades out. Sclerosis, on the other hand, has always a sharply definite outline. There is with this increase of density a change in the density which is out of proportion to the size. If a given element of the lung, for example, a lymph node, increases in size, this increase in mass must increase the density of the shadow, but if sclerotic changes are present, the degree of density is out of proportion to any increase in size which may be present. I have mentioned the places where sclerosis may occur: when such sclerosis in the lymph nodes is dense, or when the small nodes at the periphery of the hilum of the lung are involved with sclerosis, then the tuberculous origin of the sclerosis is definitely suggested. Sclerosis may occur along the bronchi with an increase in the number of bronchial markings and in the density, making them visible nearer to the periphery of the normal lung. When such sclerosis occurs along the bronchi and results in a contraction, so that the bronchial marking tends to become a straight line, instead of being irregular, it is spoken of as fibrosis. When fibrosis is present, tuberculosis is definitely suggested by this type of sclerosis. Sclerosis may occur in pulmonary infiltrations from any lesion. Of course the presence of pulmonary infiltrations is almost always an indication of previous tuberculosis. When these infiltrations are healed, they become sclerotic and leave small sclerosed nodules. These pulmonary nodules are definitely suggestive of previous tuberculosis if they occur in the infraclavicular regions, or in the regions of the upper portions of any of the lobes. These shadows of sclerosis of the lymph nodes, or the bronchial trees, or of whole pulmonary infiltrations, will serve to differentiate various classes of sclerotic changes in the lung. The first class into which I shall divide them is the normal class. The normal lymph nodes will have a degree of density which the examiner's eye must become accustomed to, so that he can evaluate any changes from that density. The normal lymph nodes are small, and their density is such that they do not stand out. The bronchial markings are few in number, they are not of any great density, they are less dense than the lymph nodes and they do not extend over the lung field any greater than two thirds of the distance to the periphery; no pulmonary nodules are present.

The second class is a group of changes which shows sclerotic changes without definitely suggesting tuberculosis. These may show sclerosis of the lymph nodes of each hilum in which there is only a moderate degree of increase of density without an increase in the size of the lymph nodes, and without a change in the small lymph nodes of the hilum. This class may include a peribronchial sclerosis in all directions about the hilum, not definitely limited to the upper lobe, not showing fibrosis and not of a marked degree.

The third class comprises cases showing sclerotic changes in such a manner as definitely suggests tuberculous origin, in which previous tuberculosis is probable. In this class are placed those cases showing the sclerotic changes, when any of the following are present: if the lymph nodes which are sclerosed are enlarged or unusually dense, or the nodes about the periphery of the hilum, are sclerosed, or a peribronchial sclerosis is present, chiefly in the upper lobes, and if fibrosis or pulmonary nodules are present, especially in the infraclavicular regions, or in the apexes of any of the lobes, or pronounced in any region.

The fourth class will include those cases in which the origin was definitely tuberculous and in which one can be absolutely assured that tuberculosis has been present previously; this class of case would include those which may be questionable in the previous classes and here are marked, with the distribution of the sclerotic changes along the bronchial trees of the upper lobes. There are other classes which comprise active conditions and which I shall not go into in the discussion.

The fifth class includes those cases in which there is a mildly active tuberculous lesion.

The sixth class includes cases in which there is a moderately active tuberculous lesion, and the seventh class is made up of cases of extensive pulmonary tuberculosis.

The class in which one would place any given case is, as I have said, subject to the personal judgment of the one who is classifying it, but it seems unlikely that any two persons would vary the classification of a given case more than one class; hence, if one finds in cases of known tuberculosis of the bones or joints or known ocular tuberculosis that the roentgenograms of the chest tend to fall into one group of classes rather than into another group, I think it shows a definite connection between a previous pulmonary lesion and tuberculosis elsewhere.

Recently, I looked over the roentgenograms of the chest of forty-nine patients that we had made at the Orthopedic Hospital, and in these forty-nine there were thirty cases which had been proved to be tuberculosis of the bones or joints, that is, proved by pathologic specimens and examinations with guinea-pigs, and every one of the thirty cases showed a chest which was placed in a class definitely indicating tuberculosis; that is, in classes from 3 to 7; in five of them there were active tuberculous conditions in the chest. Of the remaining nineteen, seven were normal, or perfectly clear, and twelve were in the first two classes of normal and those not definitely suggesting previous tuberculosis.

That is only a small group, but in a general way I find that my tendency has been to place somewhat more than 95 per cent of the cases of tuberculosis of the bones and joints, after pulmonary examinations, into classes from 3 to 7, and mostly from 4 to 7; of the nontuberculous cases, fully 90 per cent fall in classes from 1 to 3, mostly 1 and 2, as there is some overlapping in the third class. One encounters some cases of active tuberculosis in which the lesions of the bones and joints are not active tuberculosis.

I do not think I ought to attempt to show what can be seen in these pictures, because it cannot be seen except at close range, but any one who cares to examine them may do so at the end of the meeting.

DR. JAMES G. DWYER: I wish first of all to congratulate Dr. King on his excellent paper, and Dr. Derby on his discussion of it. From my own experience all I can say is that since June, 1910, for nineteen years, I have observed a series, so far as I can check them, of forty-seven patients treated with tuberculin, and I want to give you my independent analysis of tuberculin. I am aware of the history of tuberculin, back in 1880, when a lot of persons were killed by old tuberculin. I am aware that a lot of the preparations of tuberculin are imperfect agents that have been devised by bacteriologists to try to extract the true toxin from the bodies of the tubercle bacilli. I say "imperfect." Our experience is this: Tuberculin is not giving results in a lot of cases owing to two reasons: one of them is its improper use, and the other and main one is that the physicians who are using tuberculin have never bothered to study it, and learn the rationale of its use—and I am saying this from seeing men use tuberculin in my own hospital. I cannot make that too emphatic. Our experience has been that of Dr. King and Dr. Weeks.

What is meant by a cure of tuberculosis, whether it is in the lung or in the eye? We do not pretend to have a cure in the sense of a streptococcus or a staphylococcus infection. What we call a cure in tuberculosis is an arrested case.

I do not believe there is much difference between the nonexudative and the exudative types pathologically, especially since if one examines some of the so-called cases which used to be classified as nonexudative, and compares them, by means

of a slit lamp, one will get the exudate. I have seen a number of these patients treated with tuberculin, when the physician did not know whether the patient had been treated ten days or two weeks before, and I make the claim that any physician who uses tuberculin without knowing what he is doing is guilty of malpractice in one sense of the word. Tuberculin is one of the most dangerous elements that can be used, but, on the other hand, I think the weight of evidence in properly selected cases would show that tuberculin is of some therapeutic use, for example, in a patient who has lost one eye. As Dr. Derby has emphasized, every one of us who is using tuberculin makes use of every other method for the proper treatment for tuberculosis. If one treats an eye that has a tuberculous choroiditis and loses that eye, and the other eye is going, after all other kinds of treatment have failed, one must use tuberculin. I remember a woman in Trenton, N. J., who went for fourteen years without a recurrence. Spontaneous cure does not come in in my experience.

In considering the type of tuberculin, it does not matter what type one uses. The multiplicity of tuberculins means the real toxin has not yet been obtained, because with the present knowledge it cannot be. Koch extracted the old tuberculin. It was simply a 50 per cent broth extract of the tubercle bacillus, heated and diluted to 1/10 volume. Then the tubercle bacilli were ground up and extracted in salt solution, a bacillary emulsion. All these factors do not matter; the main factor is not to use large doses. We are losing sight of the truth that the cardinal fact taught for years by the bacteriologists, which we have tried to instill into the students at the College of Physicians and Surgeons was never to use tuberculin in a case in which localization was not established, and many of the cases in which treatment is given are not of that class.

I have followed some of these cases since June, 1910. A little girl was in my office last week, who for nine years had a tuberculous choroiditis of each eye. I see her every two months, and I believe she is all right. In the question of statistics, the fact that other physicians treat patients without knowing what they are doing is the reason there are so many differences. I think it is an important subject. No one claims that tuberculin is a cure, but as an adjuvant in localized tuberculosis, especially in the eye, when it is properly controlled and administered by a physician who has studied the subject, in my experience it is a wonderful aid in therapeutics.

DR. ANDREW A. EGGSTON: I see this subject from the laboratory standpoint. What I have to say is chiefly in reference to our work in the laboratory of the Manhattan Eye, Ear, Nose and Throat Hospital. Cases are referred to the laboratory by the clinician for tuberculin tests. In the past seven years we have employed the intradermal tests, using Koch's old tuberculin, and making three injections into the skin. Care is taken that 0.1 cc. of each dilution is injected directly into the epidermis. The patient returns at the end of twenty-four or forty-eight hours for a reading of the test. This test does not inconvenience the patient, and is highly sensitive and satisfactory. The subcutaneous or the Pirquet tests are not used unless specially requested. There has been a gradual increase in the number of intradermal tuberculin tests in the past seven years, as is shown in table 1.

Some physicians make the statement that almost every person will react to tuberculin. This may be true of the Pirquet test, but the statistics here given are to the contrary when the intradermal test is used. Of the 1,231 patients, 55.5 per cent had negative reactions and 44.5 per cent positive. The positive reactions are reported as +, ++, +++ and ++++, with mention of the dilutions. The tests are read in twenty-four and forty-eight hours. The time it

takes for the reaction to appear is also a guide to the extent of sensitization; in this way the clinician can get some idea of how sensitive the patient is. After testing, the patient is referred to the ophthalmologist and automatically to the social service department, and, unless otherwise ordered, he is placed on a course of doses of tuberculin, according to the schedule given in table 2. If any patient reacts poorly, treatment is stopped and the patient is referred back to the clinic. At present, 231 patients are receiving injections of tuberculin.

The ophthalmologist follows the case, but I have certain definite ideas in regard to these patients which have been formed from testing so many of them. When one encounters a patient who is sensitive to tuberculin, what does it mean? To me it simply means that the patient is sensitive to tuberculin, regardless of whether the focal process is in the eye or not. It does not mean necessarily that the patient has active tuberculosis of the eye. My general impression from the discussion is that many ophthalmologists think that it means a metastasis of tuberculosis to the eye. Personally, I do not believe it. I believe that there are two types of positive cases. It has been fairly definitely shown that in animals, when tuberculous or cured of tuberculosis, an allergy or a sensitization to tuberculin will develop, and my assumption is that a patient who reacts to tuberculin is one who has an immunity against tuberculosis. The patients who react positively to tuberculin either have an actual case of tuberculosis of the eye or an allergy

TABLE 1.—*Intradermal Tuberculin Tests in the Past Seven Years*

	Negative	Positive	Total
1922	31	27	58
1923	46	42	88
1924	91	72	153
1925	94	82	176
1926	129	98	227
1927	140	115	255
1928	152	112	264
	683, or 55.5%	548, or 44.5%	1,231

to tuberculin with lesions in the eye which may not be tuberculous. The disease process in the eye due to allergy is excited by other factors which lower the vitality of the patient, such as infections or altered metabolism. For example, in a person who is sensitive and has attacks of asthma, such as asthma caused by emanation from animals, there will come a time when that person is below par or has some infection like sinusitis or influenza, and, while free from asthma for a long time, will under the strain mentioned have an attack of asthma. I think that many of these reactions are simply local inflammatory conditions with no tubercle bacilli present, but simply manifestations of allergy, regardless of whether the inflammation is exudative or serous. This explanation accounts for some variable observations as to the value of tuberculin. In the case of allergy one will get good results. In active tuberculous nodules or actual tuberculosis of the eye, it is likely that one will get poor results, because an active infection is an entirely different process to combat; one of them is allergy, the other, actual infection due to an organism, and in that case a bacterial metastasis. From a pathologic and immunologic standpoint, there is a distinct difference in the process in which one gets an allergic reaction and that in which there is an actual infection. In the allergic cases tuberculin looks to me like the ideal protein for treatment. In the patient who is sensitive to tuberculin, the injections of tuberculin produce both a specific stimulation and a stimulation to foreign protein. Ferment reactions are stimulated more by allergic substances than by foreign pollens alone,

such as milk or serum; therefore, tuberculin is the protein to be used. Foreign proteins stimulate the general metabolism of the patient and increase the red cells and hemoglobin. This, added to the specific allergic stimulation of tuberculin, makes it the foreign protein of choice in the treatment for many inflammatory diseases of the eye. Our experience in the clinic in the Manhattan Eye, Ear and

TABLE 2.—*Schedule of Tuberculin Treatment, Bacillus Emulsion* *

Dilution 1:10,000		
Dose	Minims	Mg.
1st	3	0.0001
2d	6	0.0002
3d	9	0.0003
4th	12	0.0004
5th	15	0.0005
6th	18	0.0006
7th	21	0.0007
8th	24	0.0008
9th	27	0.0009

Dilution 1:1,000		
Dose	Minims	Mg.
10th	3	0.001
11th	6	0.002
12th	9	0.003
13th	12	0.004
14th	15	0.005
15th	18	0.006
16th	21	0.007
17th	24	0.008
18th	27	0.009

Dilution 1:100		
Dose	Minims	Mg.
19th	3	0.01
20th	6	0.02
21st	9	0.03
22d	12	0.04
23d	15	0.05
24th	18	0.06
25th	21	0.07
26th	24	0.08
27th	27	0.09

Dilution 1:1,000		
Dose	Minims	Mg.
28th	3	0.001
29th	6	0.002
30th	9	0.003
31st	12	0.004
32d	15	0.005
33d	18	0.006
34th	21	0.007
35th	24	0.008
36th	27	0.009

Dilution 1:100		
Dose	Minims	Mg.
37th	3	0.01
38th	6	0.02
39th	9	0.03
40th	12	0.04
41st	15	0.05
42d	18	0.06
43d	21	0.07
44th	24	0.08
45th	27	0.09

Dilution 1:10		
Dose	Minims	Mg.
46th	3	0.1
47th	6	0.2
48th	9	0.3
49th	12	0.4
50th	15	0.5
51st	18	0.6
52d	21	0.7
53d	24	0.8
54th	27	0.9

Dilution 1:100		
Dose	Minims	Mg.
55th	3	0.01
56th	6	0.02
57th	9	0.03
58th	12	0.04
59th	15	0.05
60th	18	0.06
61st	21	0.07
62d	24	0.08
63d	27	0.09

Dilution 1:10		
Dose	Minims	Mg.
64th	3	0.1
65th	6	0.2
66th	9	0.3
67th	12	0.4
68th	15	0.5
69th	18	0.6
70th	21	0.7
71st	24	0.8
72d	27	0.9

Continue dilution 1:10 dose 1 cc. for 1 year

* Injections should be given at three or four day intervals. This schedule is advised; however, if any severe reaction should develop, the physician will have to individualize a dosage for that particular case by decreasing the dose or lengthening the interval between injections.

Throat Hospital supports the value of tuberculin, and the majority of the attending clinicians use tuberculin with gratifying results.

DR. ERNST WALDSTEIN: When listening to the fine paper of Dr. King and the remarks of Dr. Weeks and Dr. Dwyer (only Dr. Derby struck a somewhat skeptical note), one would feel inclined to believe that treatment with tuberculin is the modern treatment for tuberculosis of the eye. Unfortunately, it has its drawbacks; it takes a long time (from forty to fifty injections being the routine

number) and ought to be repeated after a longer interval; it is by no means certain, and recurrences are rather frequent. I am certainly no opponent of the tuberculin method, and it seems to me to be superior to heliotherapy and the general tonic regimen. If, however, a clinician of such standing as Elschnig, under whose guidance some of Loewenstein's papers on tuberculosis have been published, takes refuge in such a heroic measure as a Kroenlein operation, temporary tenotomy of the externus, division of Tenon's capsule, incision of the sclera and cauterization (with some chemical agent) of a solitary tuberculous focus of the choroid (article published by Jeremija Petrovic in the *Klinische Monatsblätter für Augenheilkunde* **77**:791, 1926), the results of treatment with tuberculin cannot be so brilliant. Few of the gentlemen present would be willing to try such a drastic treatment. On account of their easy accessibility, the globe and its adnexa are evidently ideal organs for the roentgen rays and (in cases of conjunctival and corneal lesions) for treatment with radium. The risks of a permanent injury to the fibers of the lens and to the retina have certainly to be considered; however, in most cases they can be avoided if the roentgenologist is competent. I had good results from treatment with x-rays these past months in two cases in which tuberculin had failed.

DR. SIGMUND A. AGATSTON: I have had some experience with patients with general tuberculosis. I think that in the past ten years I have examined 1,000 ambulatory patients with general tuberculosis, and it seems strange to me in all these cases that I never came across a case of tuberculous or any kind of choroiditis. My impression is that the so-called, or so-recognized, tuberculosis of the choroid occurs in patients who have not any active tuberculosis; furthermore, possibly one might say, in patients who have a certain amount of resistance to tuberculosis. It is in these patients who have not any active tuberculosis that it seems to me the so-called tuberculous choroiditis is likely to occur.

DR. CLARENCE KING: It may be that Dr. Derby is right in his contention that tuberculin is valueless in its ultimate results over a period of fifteen or twenty years. It is impossible for me to draw any conclusions as to the permanent effect of tuberculin in my material over so long a time. I know that tuberculin has relieved many patients with ocular tuberculosis over a period of, at least, a few years and has enabled them to carry on their activities without interruption for long intervals. What the result may be in fifteen or twenty years, in such cases, I do not know, and should not undertake to offer predictions concerning it. I am certain that statistics relating to an indefinite number of cases similar to the personal ones described in this paper can be compiled, and that such statistics will show a large percentage of persons who have been disabled by ocular tuberculosis and who, after receiving adequate treatment with tuberculin, were at least apparently cured and who enjoyed prolonged freedom from disability. It is, of course, absolutely necessary to supplement treatment with tuberculin by all local and general methods which are likely to prove advantageous. Werdenberg has recently called attention to excellent results obtained by treatment in a sanitarium in high altitudes. Climatic treatment is clearly indicated in certain cases.

It is gratifying to hear that Dr. Dwyer has reason to believe in tuberculin. I agree with him in all that he has said as to the necessity of understanding its proper use and appreciating the safeguards which prevent the great harm which may result from its careless employment.

It was not my intention to convey the impression that general or local hypersensitiveness to tuberculin implies that the process in the eye is necessarily tuberculous. Of course, so far as the ocular lesion is concerned, only the focal reaction is of diagnostic significance. The work of Peterson on the focal reaction

is of special interest. He believes that the focal reaction in tuberculosis is partly specific and that there is also an element of intense sensitiveness of the whole body to proteins, and there is a nonspecific reaction in the neighborhood of the focus.

Dr. Eggston, I gather, maintains that in some cases in which the clinical data seem to establish the diagnosis of tuberculosis with great probability, the lesion is actually not tuberculous. Doubtless, there is a certain margin of error as is always the case when, as in tuberculosis, the diagnosis must be one of probability only. That actual tuberculosis of the eye occurs is, of course, established by the demonstration of the tubercle bacilli in the tissues by inoculation of tissue into the peritoneum of animals, and by its production by experimentation with animals.

Presumably one who is honored by an invitation to address the society is expected to present a paper which offers new facts or conclusions of value which can be objectively confirmed. I regret that I find it impossible to fulfil this requirement, as in the present state of knowledge impressions as to the value of tuberculin depend on clinical experience which has admittedly a distinctly subjective tinge.

Although not relevant, it might serve some useful purpose to mention three phases of the general subject of ocular tuberculosis which appear to me to be likely to lend themselves to original investigation and possibly to experimentation with animals: First, the focal reaction. After reading Peterson's article which embraces nontuberculous as well as tuberculous focal reactions, one realizes that we are dealing in ophthalmology in nearly all our therapy in inflammatory processes with attempts to produce a focal reaction. The second subject with possibilities is one referred to by Neumann which practically deals with the evidences of metastasis in nonpulmonary tuberculosis. It would be strange, indeed, if metastasis took place only in the eye. I am inclined to believe that between the ophthalmologist, who is interested in the eye, and the internist, who is chiefly concerned with some active evidences of tuberculosis in the body, the indefinite but significant traces of metastasis throughout the body frequently escape attention. The third phase of the subject which seemed promising was the cytotoxic influence of focal infection as a predisposing cause in the development of a tuberculous metastasis in the eye and the influence of the removal of focal infection in the cure of ocular tuberculosis.

Book Reviews

ATLAS PHOTOGRAPHISCHER BILDER DES MENSCHLICHEN AUGENHINTERGRUNDES. By the late PROFESSOR F. DIMMER and DR. ARNOLD PILLAT, Vienna. Photographs, 325. Vienna: Franz Deuticke, 1927.

Dimmer was the first to obtain satisfactory photographs of the fundus with a photographic apparatus of his own design. The subject has occupied his attention since 1906, and gradually pictures of many normal and pathologic fundi were assembled. Some of these were used to illustrate Dimmer's "Augenspiegel," third edition (Vienna: Franz Deuticke, 1921). The present collection has been made by his assistant Dr. A. Pillat for teaching purposes. About one half of the pictures were taken by Dimmer and his former assistants; the second half belong to Pillat. While Dimmer's apparatus was the only one in use, except one constructed by Zeiss, the recent camera of Nordenson, also made by Zeiss, has made the procedure more popular as the apparatus is cheaper and much simpler than Dimmer's. The author believes that this atlas will serve as an adjunct to the many excellent atlases in colors. The great advantage of the photographic representation is the possibility to show the beginning, the course and the end-stage of many processes, while usually only the more striking and typical stage is illustrated. This feature will be of value especially for teaching purposes. While one is accustomed to view the fundus in colors, after a short time pictures in black and white obtain their value especially in regard to topography of the lesion. Dr. Pillat regrets that unsolved technical difficulties make photography in colors unobtainable. In the introduction the differences between the colored picture and the photographs of the fundus are described, and the limitations of photography are also mentioned. The subject is divided into the following six parts: (1) normal fundus and congenital varieties; (2) changes of the disk; (3) changes of the optic nerve; (4) changes of the retinal vessels; (5) retinal diseases, and (6) changes of the choroid and macular region. Repeated photographs of the same process in succession, to show the course of the disease, are only one of the advantages of photography, especially as they are absolutely true to nature, and the subjective interpretation of a picture of the fundus is obviated. This is true for chorioretinitis, choked disk, hemorrhages and other conditions. Opacities of the cornea, a high degree of astigmatism, pronounced variations in level and the fact that only a limited part of the fundus is accessible are mentioned as limitations. A description of the apparatus is given. The freedom of reflexes is a special advantage of the Dimmer apparatus. The method of taking the photograph is described. The time of exposure is from one eighth to one tenth of a second, and thus the illumination causes no damage to the retina. The plates are not retouched, so that the principal value, absolute objectivity, is assured. Each plate is accompanied by an explanatory text.

A study of the plates quickly convinces one of their many good points. While the blurring and lack of colors are noticeable and dis-

turbing before one becomes accustomed to them, the topography of the whole picture, the detail of the small changes, such as in the nerve head, and the excellence of the retinal lesions must be apparent even to the most critical observer. It is clear that a great many more pictures can be shown by photography, so that the author's real contribution by this atlas, as a supplement to the previous atlases, is undoubtedly true. The labor and the many painstaking trials which the development of this method must have cost are the usual attribute, but also the reward of any difficult pioneer investigation, and whatever the future holds in store in the development of the graphic arts, all credit belongs to the late lamented, talented and modest Dimmer. For the completion and publication of these investigations, ophthalmology is indebted to Dr. Pillat.

A. K.

GRUNDRISS DER AUGENHEILKUNDE. A Textbook of Ophthalmology for Students and Practitioners. By PROFESSOR A. BRÜCKNER, Director of the University Eye Clinic, Basel, and PROFESSOR W. MEISSNER, Director of the University Eye Clinic, Greifswald. Price, 25 marks. Second enlarged edition. Pp. 600, with 221 illustrations and 9 colored plates. Leipzig: Georg Thieme, 1929.

The authors have attempted a middle course between the large and expensive textbooks and the small compends which exclude scientific educational purposes. The number of illustrations was kept in narrow bounds by the belief that the best illustrations even in color do not replace clinical experience. The subject matter is treated in a somewhat different manner from the usual, and the relation between diseases of the eye and general diseases is emphasized. Anomalies of refraction are treated for the first time on the basis of Gullstrand's teachings. In this, the second edition (the first appeared in 1920), the scientific achievements of the last decade are added, and the subject matter is brought well up to date. The first part is devoted to normal anatomy, embryology, physiology, inheritance and developmental defects. The second part (special pathology) treats of diseases of each anatomic part, errors of refraction and accommodation, glaucoma and injuries. In the third part the methods of examination are described, with chapters on bacteriology and serology. Finally, treatment is described in part four, which includes operations; a chapter on blindness and the care of the blind is added. A list of prescriptions and an index complete the volume.

A survey of the book quickly convinces one that this is not an elementary book, but a complete and scholarly treatise which can with profit be studied by the advanced student and by the ophthalmic practitioner. It reflects the high scientific character of the German ophthalmology of the present day. The illustrations are well selected and are well executed. In many cases the description of the conditions includes an illustrated explanation of the pathologic lesion. The illustrations in color show the bacteria, the lesions of the lids, conjunctiva and iris and the more usual changes of the fundus. These are new and well done.

A. K.

Directory of Ophthalmologic and Otolaryngologic Societies *

FOREIGN

OXFORD OPHTHALMOLOGICAL CONGRESS

Master: Dr. A. B. Cridland, Salisbury House, Chapel Ash, Wolverhampton. England.
Secretary: Dr. C. G. Russ-Wood, 12 St. John's Hill, Shrewsbury, England.
Place: Oxford, England. Time: July 4-5, 1929.

INTERNATIONAL

INTERNATIONAL CONGRESS OF OPHTHALMOLOGY

President: Dr. E. Treacher Collins, 17 Queen Anne St., London, England.
Secretary: Dr. E. Mark, Academisch Zickenhuis, Leyden, The Netherlands.
Place: Amsterdam, The Netherlands. Time: Sept. 5-13, 1929.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Edward B. Heckel, Jenkins Arcade Bldg., Pittsburgh.
Secretary: Dr. William C. Finnoff, Imperial Bldg., Denver.
Place: Portland, Ore. Time: July 10-12, 1929.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Harris P. Mosher, 828 Beacon St., Boston.
President-Elect: Dr. William H. Wilder, 122 S. Michigan Ave., Chicago.
Executive Secretary: Dr. William P. Wherry, Medical Arts Bldg., Omaha.
Place: Atlantic City, N. J. Time: Oct. 21-25, 1929.

AMERICAN OPHTHALMOLOGICAL SOCIETY

President: Dr. Walter R. Parker, David Whitney Bldg., Detroit.
Secretary: Dr. Emory Hill, 501 E. Franklin St., Richmond, Va.
Place: Hot Springs, Va. Time: June 10-12, 1929.

LOCAL

LOS ANGELES COUNTY MEDICAL SOCIETY, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Harold S. Muckleston, 6422 Hollywood Blvd., Los Angeles.
Secretary-Treasurer: Dr. J. Frank Friesen, 1208 Roosevelt Bldg., Los Angeles

MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA, SECTION OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

Chairman: Dr. Walter A. Wells, 1606 20th St. N. W., Washington.
Secretary: Dr. J. N. Greear, Jr., 1740 M St. N. W., Washington.
Place: 1718 M St. N. W. Time: 8 p. m., third Friday of each month.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

ATLANTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. H. F. McDuffie, 45 Edgewood Ave., Atlanta, Ga.
Secretary: Dr. Leon E. Brawner, 157 Forrest Ave. N. E., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St. N. E. Time: 8 p. m., last Thursday of each month.

INDIANAPOLIS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Lafayette Page, Hume-Mansur Bldg., Indianapolis.
Secretary: Dr. Kenneth L. Craft, Hume-Mansur Bldg., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

NEW ORLEANS OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. M. Earle Brown, Maison Blanche, New Orleans.
Secretary-Treasurer: Dr. W. A. Wagner, Pere Marquette Bldg., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital, New Orleans. Time: 8 p. m., third Thursday of each month from September to June.

BALTIMORE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Leon J. Goldbach, 1012 N. Charles St., Baltimore.
Secretary: Dr. Angus Lloyd MacLean, Johns Hopkins Hospital, Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St., Baltimore. Time: 8:30 p. m., fourth Thursday of each month from October to May.

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. W. Holbrook Lowell, 82 Commonwealth Ave., Boston.
Secretary-Treasurer: Dr. E. B. Dunphy, 520 Commonwealth Ave., Boston.
Place: Massachusetts Eye and Eye Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

DETROIT OPHTHALMOLOGICAL CLUB

Chairman: Members rotate alphabetically.
Secretary: Parker Heath, David Whitney Bldg., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

KANSAS CITY SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. L. R. Forgrave, Logan Bldg., St. Joseph, Mo.
Secretary: Dr. A. E. Eubank, Argyle Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

ST. LOUIS OPHTHALMIC SOCIETY

President: Dr. William H. Luedde, Metropolitan Bldg., St. Louis.
Secretary: Dr. Leo L. Mayer, Carleton Bldg., St. Louis.
Place: St. Louis or Washington University Cafeteria.
Time: 6:30 p. m., fourth Friday of each month from October to April, inclusive.

OMAHA AND COUNCIL BLUFFS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. Charles M. Swab, Acquila Court Bldg., Omaha.
Secretary: Dr. J. C. Davis, 1615 Howard St., Omaha.
Place: Medical Arts Tea Room. Time: 5:15 and 7:00 p. m. Third Wednesday of each month from October to May.

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Lee W. Hughes, 1019 Broad St., Newark.
Secretary: Dr. Earle LeRoy Wood, 31 Lincoln Park, Newark.
Place: 91 Lincoln Park South. Time: 8:45 p. m., second Monday of each month.

BROOKLYN OPHTHALMOLOGICAL SOCIETY

President: Dr. Robert M. Rogers, 680 St. John's Pl., Brooklyn.
Secretary: Dr. William F. C. Steinbugler, 515 Ocean Ave., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave., Brooklyn.
Time: Third Thursday of February, April, May, October and December.

EASTERN NEW YORK EYE, EAR, NOSE AND THROAT ASSOCIATION

President: Dr. M. S. Lord, 148 Barrett St., Schenectady.
Secretary-Treasurer: Dr. Arthur F. Holding, 142 Washington Ave., Albany.

BUFFALO OPHTHALMOLOGIC CLUB

President: Dr. E. E. Blaauw, 190 Ashland Ave., Buffalo.
Secretary-Treasurer: Dr. A. F. Luhr, Medical Arts Bldg., Buffalo.

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman: Dr. B. Samuels, 57 W. 57th St., New York.
Secretary: Dr. T. B. Johnson, 30 W. 59th St., New York.
Time: 8:30 p. m., third Monday of every month.

ROCHESTER EYE, EAR, NOSE AND THROAT SOCIETY

Chairman: Dr. Frank Barber, 75 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. Cecil B. Hert, 29 N. Goodman St., Rochester, N. Y.
Place: Rochester Medical Assn., 113 Prince St. Time: 8 p. m., third Monday of each month from October to May.

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. George M. Constans, Bismarck.
Secretary-Treasurer: Dr. F. L. Wicks, Valley City.

CLEVELAND ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

Chairman: Myron Metzenbaum, Rose Bldg., Cleveland.
Secretary: Dr. A. D. Ruedemans, Euclid and E. 93d St., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Joseph L. McCool, 909 Stevens Bldg., Portland.
Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

THE PITTSBURGH OPHTHALMOLOGICAL SOCIETY

President: Dr. Edward B. Heckel, Jenkins Arcade Bldg., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg., Pittsburgh. Time: Fourth Monday of each month, except June, July, August and September.

THE PITTSBURGH SLIT-LAMP SOCIETY

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: 121 University Pl., Pittsburgh. Time: Second Monday evening of each month, except June, July, August and September.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. Robert R. Hampton, Boston Bldg., Salt Lake City.
Secretary-Treasurer: Dr. Walter F. Hoffman, 817 Summit Ave., Seattle.
Place: Salt Lake City: Time: July 1 and 2, 1929.

COLLEGE OF PHYSICIANS, PHILADELPHIA, SECTION ON OPHTHALMOLOGY

Chairman: Dr. C. E. G. Shannon, 17th and Walnut Sts., Philadelphia.
Clerk: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Time: Third Thursday of every month.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. William Thornwall Davis, 927 Farragut Sq., Washington, D. C.
Secretary: Dr. Fletcher D. Woodward, Box 162, University, Va.
Place: Miami, Fla. Time: Nov. 20-22, 1929.

THE MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Rychener, Exchange Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. John G. McLaurin, Medical Arts Bldg., Dallas, Texas.
Executive Secretary: Dr. W. Mood Knowles, Medical Arts Bldg., Dallas, Texas.
Recording Secretary: Dr. L. M. Sellers, Medical Arts Bldg., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

FORT WORTH EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. C. P. Schenck, Medical Arts Bldg., Fort Worth, Texas.
Secretary-Treasurer: Dr. R. H. Needham, Fort Worth National Bank Bldg., Fort Worth, Texas.
Place: University Club. Time: 6:30 p. m., first Friday of each month except July and August.

HOUSTON ACADEMY OF MEDICINE, EYE EAR, NOSE AND THROAT SECTION

President: Dr. John H. Foster, 1300 Walker Ave., Houston, Texas.
Secretary: Dr. Edward W. Griffey, Houston Clinic, Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., first Thursday of each month from September to June.

THE SAN ANTONIO OPHTHALMOLO-OTO-LARYNGOLOGICAL SOCIETY

President: Dr. F. H. Roseborough, 302 Moore Bldg., San Antonio, Texas.
Secretary-Treasurer: Dr. William D. Gill, 323 Medical Arts Bldg., San Antonio, Texas.
Place: Bexar County Medical Library, San Antonio, Texas. Time: 8 p. m., first Tuesday of each month from October to May.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Will Otto Bell, Medical-Dental Bldg., Seattle.
Secretary-Treasurer: Dr. Francis A. Brugman, Medical-Dental Bldg., Seattle.
Time: 8 p. m., third Tuesday of each month, except July and August.

NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Herschel Ezell, Doctor's Bldg., Nashville, Tenn.
Secretary-Treasurer: Dr. W. W. Wilkerson, Jr., Bennie-Dillon Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month.

VIRGINIA SOCIETY OF OTO-LARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. H. B. Stone, Roanoke.
Secretary-Treasurer: Dr. Fletcher D. Woodward, Box 162, University.

SPOKANE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. O. M. Rott, Paulsen Bldg., Spokane Wash.
Secretary: Dr. C. A. Veasey, Jr., Spokane, Wash.

THE MILWAUKEE OTO-OPHTHALMIC SOCIETY

President: Dr. L. P. Allen, 145½ Main St., Oshkosh, Wis.
Secretary-Treasurer: Dr. Edward R. Ryan, 425 E. Water St., Milwaukee.
Place: University Club. Time: 6:30 p. m., third Tuesday of each month.

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